VOLUME 42 JULY, 1939 NUMBER 1

[FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY]

LIBRARY OF CONGRESS AUG 18 1939

The AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

OFFICIAL ORGAN
THE AMERICAN ROENTGEN RAY SOCIETY
THE AMERICAN RADIUM SOCIETY

Editor: LAWRENCE REYNOLDS, M.D.

Editorial Board

A. C. CHRISTIE, M.D. E. H. SKINNER, M.D. LAURISTON S. TAYLOR

Advisory Board for Pathology

JAMES EWING, M.D. EUGENE L. OPIE, M.D.

ISSUED MONTHLY FOR THE AMERICAN ROENTGEN RAY SOCIETY
BY CHARLES C THOMAS, PUBLISHER, SPRINGFIELD, ILLINOIS

AMERICAN ROENTGEN RAY SOCIETY
Fortieth Annual Meeting
CHICAGO, ILLINOIS · SEPTEMBER 19-22, 1939

AMERICAN RADIUM SOCIETY
Twenty-fifth Annual Meeting
NEW YORK CITY · 1940

YEARLY SUBSCRIPTION, $10.00 SINGLE NUMBER, $1.50

X-RAY SHADOWGRAPHS

Advertisement No. 3 in a series describing interesting non-medical uses of Du Pont X-ray Film

Above: Photo of portrait before cleaning
Below: Photo of portrait after cleaning

X-RAY SHADOWGRAPH

X-RAY DIAGNOSIS = "A Two-faced Lady"

WELL known is the fact that art critics use x-ray "shadowgraphs" to discover forgeries and old paintings beneath subsequent alterations. One so utilized by The Metropolitan Museum of Art disclosed the original face on the portrait by Frans Pourbus illustrated above. The cross-bars on the "shadowgraph" are produced by the wooden bracing on the back of the portrait.

NOT so well known is the fact that x-ray "shadowgraphs" also are used to analyze the technique of the Old Masters, their brush work, their materials, even the changes they made in the canvas while painting it.

Naturally to x-ray a thin coat of paint requires a film with extremely fine gradation. Whether you are looking for faces or fractures, for the maximum amount of detail use Du Pont X-ray Film.

DU PONT X-RAY FILM

DU PONT FILM MANUFACTURING CORPORATION, INC. 9 ROCKEFELLER PLAZA, NEW YORK, N.Y.

FOR BETTER RADIOGRAPHS

AUG 10 1939

©CIB 425374

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 42 JULY, 1939 NO. 1

CONTENTS

Determination of Heart Size . . . FRED JENNER HODGES, M.D. 1

Visualization of the Chambers of the Heart, the Pulmonary Circulation, and the Great Blood Vessels in Heart Disease . . . . . GEORGE P. ROBB, M.D., AND ISRAEL STEINBERG, M.D. 14

Inflammatory Diseases of the Pericardium . EUGENE FREEDMAN, M.D. 38

Roentgenological Aspects of Acquired Valvular Heart Disease . . . . . MERRILL C. SOSMAN, M.D. 47

Roentgenologic Aspects of Non-valvular Disease of the Heart . . . . . GEORGE LEVENE, M.D. 60

Roentgenology of Congenital Cardiovascular Disease HUGO ROESLER, M.D. 72

Roentgen Examination of the Aorta and Pulmonary Artery . . . . . MARCY L. SUSSMAN, M.D. 75

Persistent Vertebral Process Epiphyses . . WILBUR BAILEY, M.D. 85

Motion in the Vertebral Column . . . . JOSEPH F. ELWARD, M.D. 91

Fractures of the Sacrum; Their Incidence in Fracture of the Pelvis . . . . . J. P. MEDELMAN, M.D. 100

Congenital Pseudarthrosis of the Tibia . . CLIFTON R. SCOTT, M.D. 104

Physiological Effects of Radiation: III. The Lethal Effect of Roentgen Radiation Produced by Various Kilovoltages (50 to 1000 kv.) upon the Brown-Pearce Rabbit Epithelioma . . . . . ROGER A. HARVEY, . . RICHARD DRESSER, M.D., AND STAFFORD L. WARREN, M.D. 110

The Relation between Tin and Copper Filtration when Using a Thick-Walled Roentgen Tube . . . . . M. C. REINHARD AND H. L. GOLTZ 122

Editorials

The Annual Meeting 126

Byron Hubbard Jackson: 1873–1939 127

Society Proceedings, Correspondence and News Items 130

Book Reviews 138

Department of Technique

A Device for Maintaining Developing Solutions at a Constant Temperature . . . PAUL A. BISHOP, M.D., AND GEORGE E. BIERCE, E.E. 141

Stereoroentgenoscopy Using Polarization . . WILLIAM SNOW, M.D. 143

Abstracts of Roentgen and Radium Literature 145

GENERAL INFORMATION

Manuscripts offered for publication and other correspondence relating to the editorial management and books for review should be sent to Dr. Lawrence Reynolds, Editor, 110 Professional Bldg., Detroit, Michigan. Contributions may be sent in foreign languages and will be translated if found acceptable. Original articles are published only with the understanding that they are contributed exclusively to this JOURNAL.

A certain number of illustrations will be supplied free of cost; but special arrangements must be made with the Editor and Publisher for excess illustrations and elaborate tables.

Fifty reprints are furnished without charge to authors. Additional reprints may be purchased according to a scale of prices that accompanies galley proofs.

For information regarding membership in the American Roentgen Ray Society, address the Chairman of the Executive Council, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio.

For information regarding the program of the annual meeting, address the President-Elect, Dr. E. L. Jenkinson, St. Luke's Hospital, Chicago, Illinois.

Correspondence regarding subscriptions, advertisements, and other business relating to THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY should be addressed to Charles C Thomas, Publisher, 220 East Monroe Street, Springfield, Illinois. Foreign subscriptions may be addressed to Baillière, Tindall & Cox, 7 and 8, Henrietta Street, Covent Garden, London, W. C. 2, England; H. K. Lewis & Co., Ltd., 136, Gower Street, London, W. C. 1, England; Maruzen Co., Ltd., 11–16 Tori-Sanchome, Nihon-bashi-Ku, Tokyo, Japan; Beutelspacher & Cia., Sarmiento 815, Buenos Aires, Argentina; W. Watson & Sons, Ltd., Watson House, Bligh Street, Sydney, Australia; or to your own agent.

Subscription price a year: $10.00, United States; $11.00, Canada; $12.00, other countries. Current single numbers, $1.50.

SUGGESTIONS FOR PREPARATION OF MANUSCRIPTS

Manuscripts should be typewritten, with double spacing, and good margins (not on thin paper) and the *original* should be sent to the Editor. The author should keep a carbon copy, as the original is not returned. The name of the author should appear on each sheet of manuscript. Tabular materials should be typed on separate sheets; likewise bibliographical lists, footnotes, etc.

Drawings and charts for reproduction should be made in black (*never* in blue). Photographic prints of plates or slides on glossy paper produce the best half-tones.

All photographs and drawings intended for illustrations should be numbered, the top plainly indicated, and an abbreviated title of the paper placed on the back of *each one*.

Legends for illustrations should be typewritten in a single list, with numbers corresponding to those on the photographs and drawings, instead of attaching each legend to its corresponding picture.

Care in preparation of bibliographies will save much time and correspondence. Each reference in a bibliography should include the following information in the order indicated: Name of the author with initials; title of the article; name of the periodical; year, volume, and page. It is requested that the authors use the following as a model:

1. Gardner, W. J., and Frazier, C. H. Ventriculography without air injection. J. Am. M. Ass., 1929, 93, 193–194.

The month should be given if the reference is to an issue of the current year. An alphabetical arrangement of authors is preferred.

The author should always place his full address somewhere on his manuscript. This is very important and is often omitted.

The American Journal of Roentgenology and Radium Therapy is owned, maintained by, and in the interests of, the American Roentgen Ray Society, Inc. It is issued monthly by Charles C Thomas, Publisher, 220 East Monroe Street, Springfield, Illinois.

The American Journal of Roentgenology and Radium Therapy does not hold itself responsible for any statements made or opinions expressed by any contributors in any article published in its columns.

Entered as second-class matter February 10, 1930, at the postoffice at Springfield, Illinois, and Menasha, Wisconsin, under the Act of March 3, 1879. Accorded the special rate of postage provided for in paragraph 4, section 412, P.L. and R.

Copyright 1939 by American Roentgen Ray Society, Inc. All Rights Reserved. This issue may not be reproduced, in whole or in part, in any form (except by reviewers for the public press) without written permission from the publishers.

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 42 JULY, 1939 No. 1

DETERMINATION OF HEART SIZE*

By FRED JENNER HODGES, M.D.
ANN ARBOR, MICHIGAN

THE voluminous literature in the field of cardiac roentgenology is devoted to a very considerable extent to the subject of cardiac measurement. There has been a consistent effort on the part of investigators to devise a trustworthy method of determining the true size of the heart in individual subjects, and to determine whether such measured size is normal or abnormal for the individual in question. This ambitious aim has not, as yet, been realized nor is it apt to be if one insists upon mathematical accuracy. Since the heart is a geometrical solid which cannot during life be subjected to displacement methods for the measurement of volume, the plan of substituting obtainable measurements in formulae which express volume must be employed. Unfortunately, the human heart does not conform strictly to the shape of any geometrical solid for which a simple and universally applicable volume formula can be derived. All methods thus far proposed represent estimations of volume from available diameter or area measurements in one or more planes, or merely derivations of indices of size which can be substituted for volume. Prediction tables of normal heart size based upon reasonably well correlated and easily obtainable criteria, such as age, sex, height, weight, chest girth, body surface area, etc., however carefully prepared, are at best expressions of statistical probability to which biological forces cannot be held strictly accountable. It is possible to estimate but not to determine normal cardiac size for a given individual.

Notwithstanding the ingenuity with which the problem has been attacked by numerous investigators, an imposing array of technical difficulties related to the anatomy and the physiology of the circulatory apparatus persists in preserving the secret of exact cardiac size during life. Regardless of projection angle, it is quite impossible to observe by roentgen methods all segments of the cardiac contour. Except in unusual pathological situations, one cannot differentiate between the shadows produced by the heart itself and the great vessels at their points of attachment. In nearly every human subject, a considerable portion of the cardiac surface lies in close contact with the diaphragm, rendering the heart contour invisible in this zone. Movements of the heart throughout its own cycle and during the cycle of respiration are extremely complex. The volume of the entire heart as well as the volumes of its individual chambers undergo rapid and complex changes. During the Valsalva ef-

* From the Department of Roentgenology, University of Michigan, Ann Arbor, Michigan. Read in a Symposium on Heart Disease at the Thirty-Ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938.

fort, in which intrathoracic pressure is increased, the heart becomes noticeably smaller, while during the Müller exercise, circumstances are reversed. Diastolic cardiac volume varies considerably according to pulse rate. Posture and habitus are clearly related to the position of the heart within the thorax. Even at autopsy exact measurement of the heart volume at the moment of death is defeated by the prosector's inability to cope successfully with such factors as volume changes associated with rigor mortis, terminal dilatation of the right heart, the almost inescapable loss of blood attendant upon the manipulation necessary to remove the heart from the chest.

Although the normal position of the heart during life cannot be reliably described with anatomical accuracy because of its normal mobility, certain relationships are relatively constant and should be clearly appreciated. As viewed in the anteroposterior axis of the thorax, the main mass of the heart lies somewhat to the left of the midline, the aorta and the pulmonary artery arise to the right and at the midline respectively and extend upward and toward the left. The cardiac apex is directed, in general, downward and toward the left and at the same time toward the chest wall anteriorly. As seen in this projection, the right border of the cardiac silhouette represents the right auricle from the diaphragm level to a point near the third rib level where it is overlapped by the prominent outline of the ascending aorta and the less clearly visible shadow of the superior vena cava. The left border of the silhouette represents the margin of the left ventricle overlapped for a short distance immediately beneath the prominent bulge of the pulmonary artery by a small portion of the left auricular appendage. Above the bulge produced by the pulmonary artery can be seen the "knob" of the aorta projecting to the left of the dense vertebral shadow just beneath the clavicles.

Because of the fact that distortion is inevitable when an object is projected in a beam of divergent rays, it is important to take steps to minimize this distortion in preparing silhouettes intended for use in estimating size. The mean plane of the heart ordinarily lies 6–8 cm. posterior to the anterior chest surface. Therefore, distortion can be best controlled by preparing silhouettes in posteroanterior projection. In roentgen examination of the heart, it is customary to employ this type of projection at the relatively long target-film distance of 2 meters, or in this country, 6 feet. Roentgenograms so produced are known as teleroentgenograms.

Viewed in the direct transverse axis of the chest, the heart is inclined from its base toward its apex from above downward. The cardiac apex itself lies in close apposition to the anterior chest wall. The aorta arising from the base of the left ventricle curves in a smooth arc to the anterior margin of the vertebral column, beyond which it passes into the sulcus at the left. In normal subjects during quiet respiration the widest point of the posterior cardiac surface is separated by 3–4 cm. from the vertebral column. As seen in this projection the anterior contour of the silhouette represents the left ventricle below, the right ventricle at the base of the pulmonary artery above, while the posterior contour represents left ventricular margin below and the posteriorly placed left auricle above. The division point between these latter structures is not well defined in the normal heart, but in well-established mitral stenosis specific enlargement of the left auricle can be demonstrated to good advantage in the direct lateral projection.

Two other projections are commonly used in roentgen examination of the heart because they afford opportunity to observe features which cannot be appreciated in either the frontal or direct lateral positions. When the chest is viewed in the 45 degree right anterior oblique position, the heart appears to be displaced toward the left of the spine and hitherto invisible portions of the silhouette come into view.

This projection is accomplished by positioning the patient with face toward the film or screen at an angle of 45 degrees, the right shoulder close to the recording surface, the arm at the side, the left arm resting on the head. Under these circumstances, the right border of the silhouette represents the overlapped margins of right and left auricles together with the junction of the great veins which empty into them. The base of the aorta is responsible for the uppermost portion of its contour. The left margin of the silhouette which now appears elongated laterally to some extent represents the right ventricle, the origin and the distal portions of the pulmonary artery. The left ventricle is now concealed posteriorly. This projection corresponds almost exactly with the plane bounded by the arch of the aorta and consequently affords an opportunity to observe the caliber of this vessel.

The exact reverse of this position, known as the left anterior oblique, yields a cardiac silhouette of quite different appearance. The heart now appears to be displaced toward the right of the vertebral column, and the aortic arch sweeps from right to left, almost exactly perpendicular to the angle of vision. The observer is now looking almost directly at the apex of the heart and, as a result, the cardiac silhouette appears to be nearly circular in shape and smaller than when seen in the opposite oblique. Its right margin represents the border of the right ventricle; the left margin represents the left ventricle. The auricles are almost completely hidden in this view. Schematic diagrams developed upon direct tracings from teleroentgenograms so prepared are shown in Figure 1.

The roentgenoscopic method of obtaining cardiac silhouette tracings, if properly carried out, may be substituted for the roentgenographic procedure of teleroentgenography. The underlying principle of obtaining cardiac silhouettes with little or no distortion is the same. Orthodiagraphy eliminates distortion by employing a narrow beam and recording individual points along the contour of the cardiac shadow as they are projected by a perpendicular pencil of rays. The method actually represents piecemeal parallel ray projection. Inexperienced observers are apt to have difficulty with this procedure, although with a little practice its technique can easily be mastered. In actual practice, roentgenoscopy and roentgenography are commonly combined in every thoroughgoing examination of the heart. If one's roentgenoscopic equipment is designed to permit orthodiagraphic procedures, it is convenient at times to check roentgenographic measurements against those obtained by orthodiagraphy, and occasionally it will be found that only in this manner can satisfactory cardiac silhouettes be obtained.

Four methods of approaching the matter of heart size determination in common clinical usage are illustrated in Figure 2. All of the silhouettes here shown have been traced directly from the clinical record of a female patient (E.G., reg. 375563, aged fifty-three, height 64 inches, weight 129 lb., pulse rate at the time of examination 126 per minute). The most widely used procedure of heart measurement consists of determining standard frontal diameters. Three points are located along the right border of the silhouette, the junction of the great vessels and the right auricle, the widest point of the silhouette on the right, and the junction of the heart border and the diaphragm below; and three points on the left margin, the junction of left auricular appendage and pulmonary artery, the widest point of the silhouette on the left margin, and the junction of heart shadow and diaphragm below. From these are constructed the long axis of the heart, the broad axis measured perpendicularly to this line, and the greatest transverse diameter. Tables are available for comparing the values obtained against probable normals. In addition, the greatest transverse diameter can be compared against the transverse thoracic diameter measured at the level of the cardiac apex from the inner surfaces of the ribs on either side.

This chest diameter divided by the greatest width of the heart yields the cardiothoracic ratio. Quotients materially less than 2.0 are considered to indicate cardiac enlargement. from the inner margins of the ribs on the right to the tips of the transverse vertebral processes on the left, is used as an index of relative heart size. The normal value of

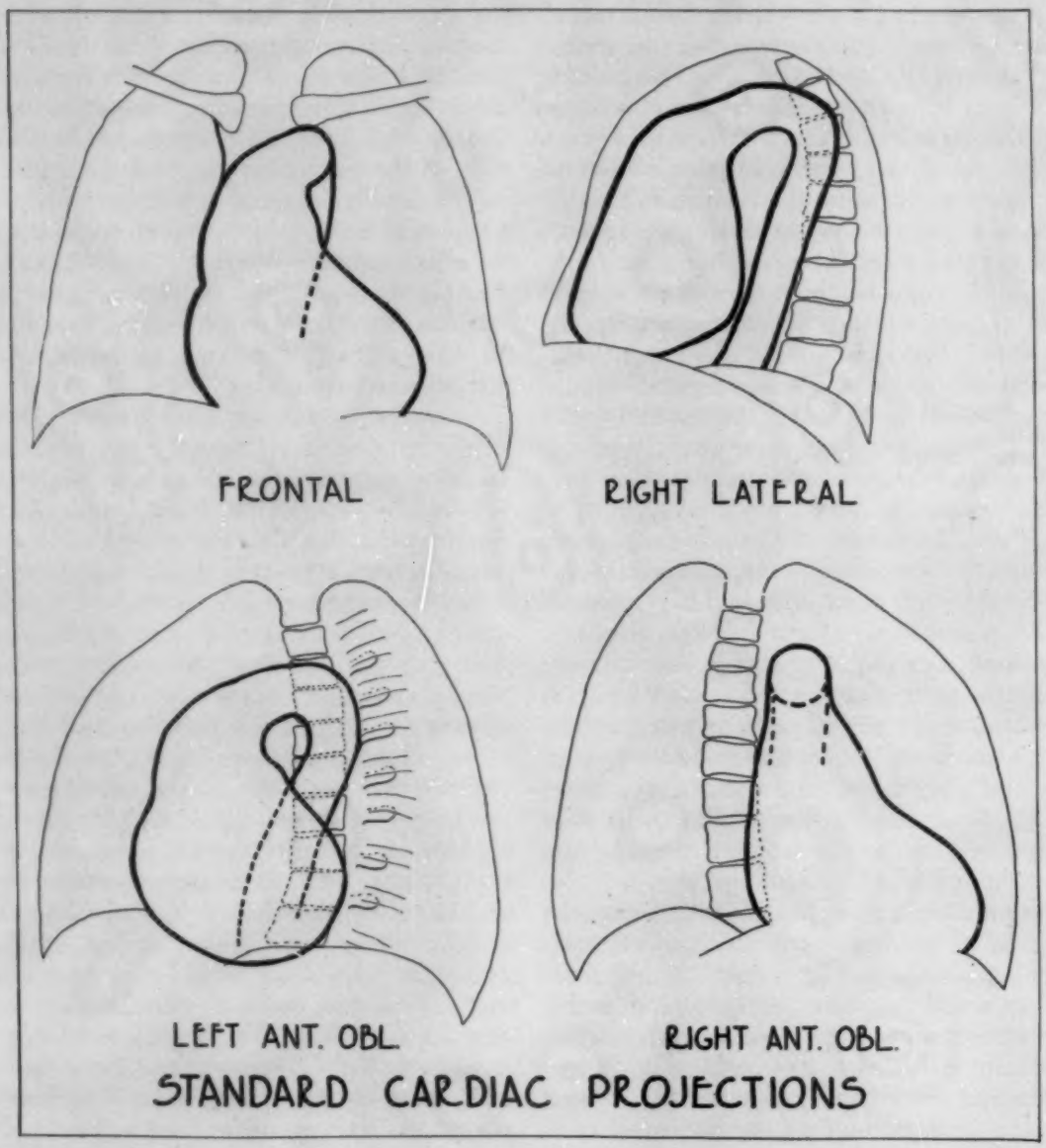


Fig. 1

In the case here used for illustration, this ratio is normal.

Employing a silhouette made in the left anterior oblique projection, the greatest diameter of the heart compared with an arbitrary oblique chest diameter, taken this ratio is 2.0. In the case in question, the value 1.6 is obtained, which falls definitely within the range of pathological enlargement. By this same method of approach it is possible, by measuring the horizontal distance from the inner surface of the chest

wall at the level of the carina to the right margin of the trachea, laying off a similar distance to the left and observing where a point so located will lie with respect to the

the silhouette are assumed to represent the relative diameters of the right and left ventricles, respectively. If one-fourth of the oblique thoracic diameter above de-

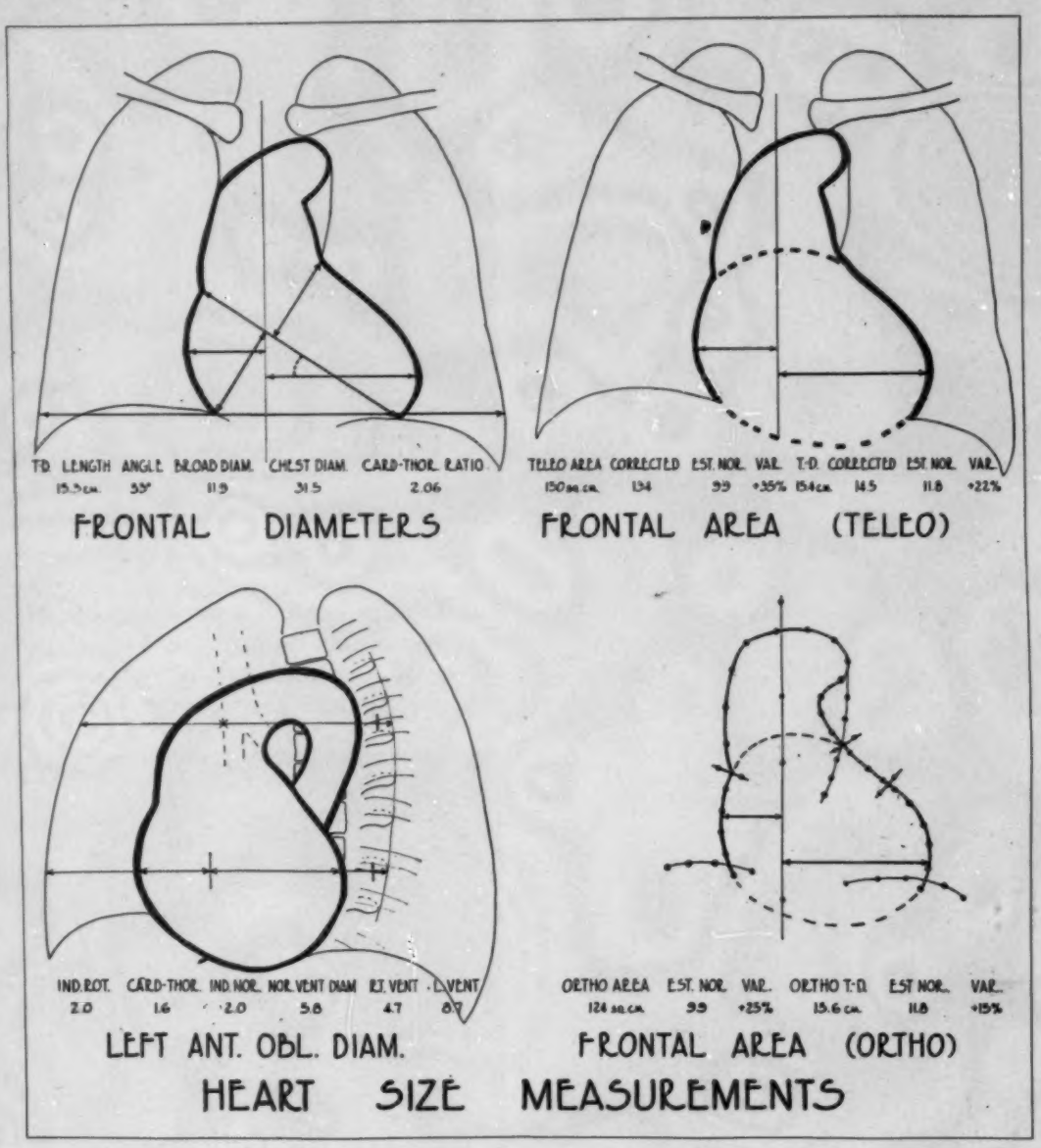

T.D. LENGTH ANGLE BROAD DIAM. CHEST DIAM. CARD-THOR. RATIO
15.5 cm. 35° 11.9 31.5 2.06
FRONTAL DIAMETERS
TELEO AREA CORRECTED EST. NOR. VAR. T-D. CORRECTED EST. NOR. VAR.
150 sq. cm. 134 99 +35% 15.4 cm. 14.5 11.8 +22%
FRONTAL AREA (TELEO)
IND. ROT. CARD-THOR. IND. NOR. NOR. VENT DIAM. EST. VENT L. VENT
2.0 1.6 2.0 5.8 4.7 8.7
LEFT ANT. OBL. DIAM.
ORTHO AREA EST. NOR. VAR. ORTHO T-D. EST. NOR. VAR.
124 sq. cm. 99 +25% 13.6 cm. 11.8 +15%
FRONTAL AREA (ORTHO)
HEART SIZE MEASUREMENTS

Fig. 2

visible ends of the transverse processes, to locate at the midpoint on a similar horizontal line at the broadest portion of the cardiac shadow the location of the interventricular septum. Measurements from this point to the right and left margins of

scribed is assumed to represent the average diameter of each ventricle, values above this assumed normal may be interpreted as evidence of abnormal enlargement. According to this rather complicated and distinctly empirical method of computa-

Fig. 3

Fig. 5

tion, it is found that in the case in question the left ventricular diameter is distinctly above normal.

Instead of depending upon linear dimensions for purposes of evaluating heart size, one may choose to measure projected cardiac area, in general a more reliable index of volume. To do so, the frontal plane silhouette is completed by extending the curved right and left margins, and with a

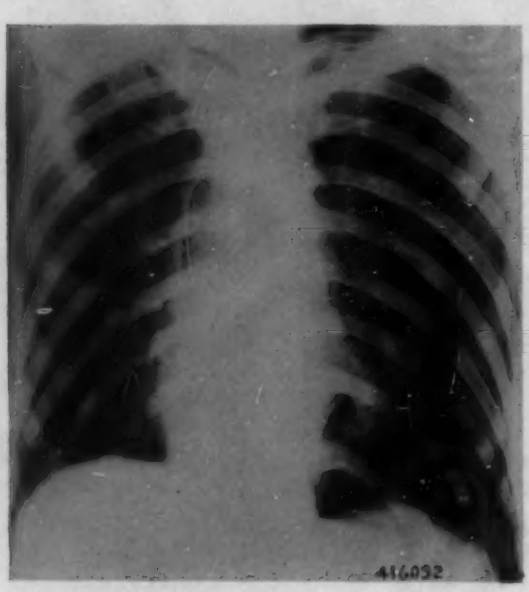


Fig. 4

Fig. 6

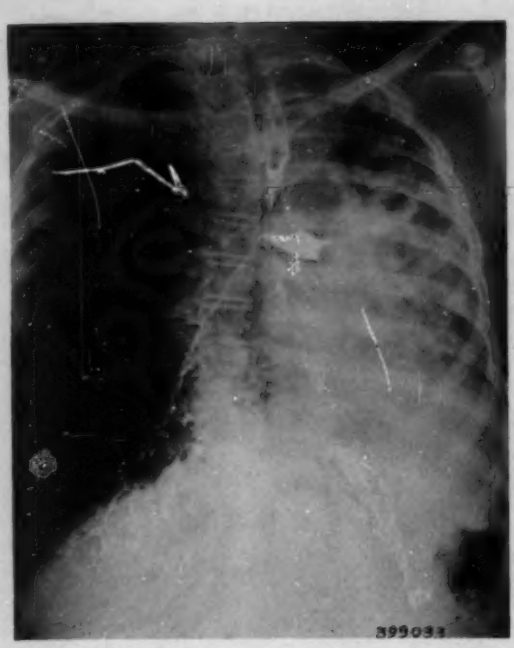


Fig. 7

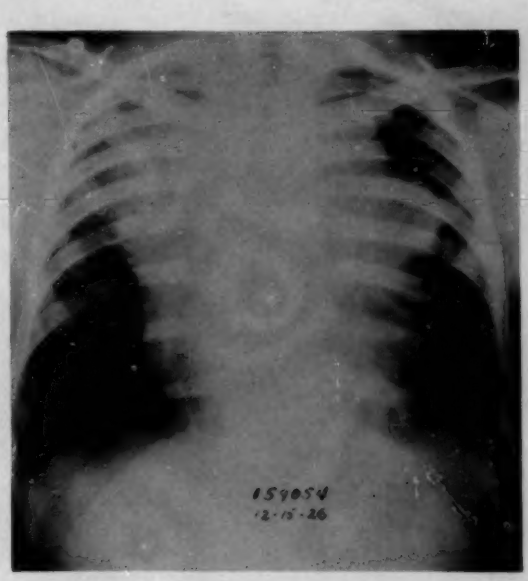


Fig. 9

draftsman's planimeter, designed to read in square centimeters, the enclosed area is determined. When corrected for inherent distortion, this value may be compared with the estimated normal based on age, height and weight available in convenient tabulated form. The final results are ex-

Fig. 8

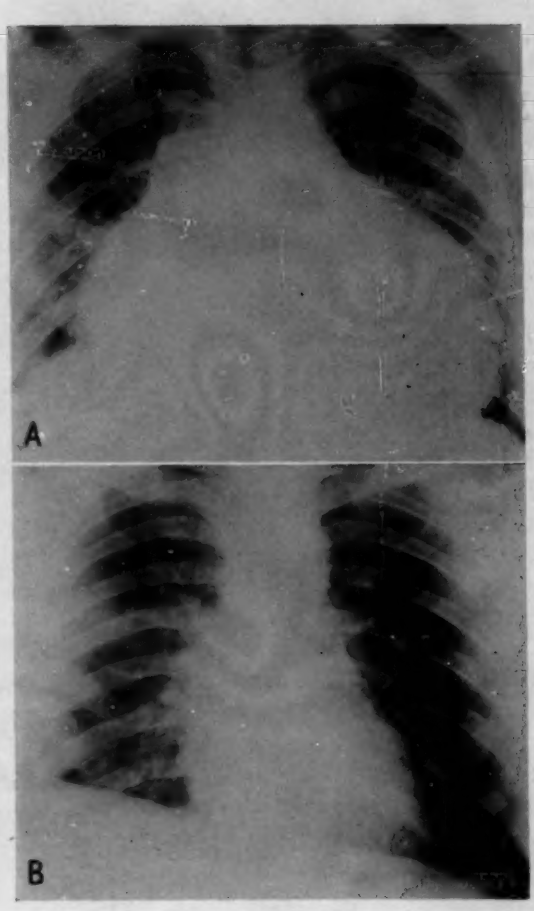


Fig. 10, *A* and *B*.

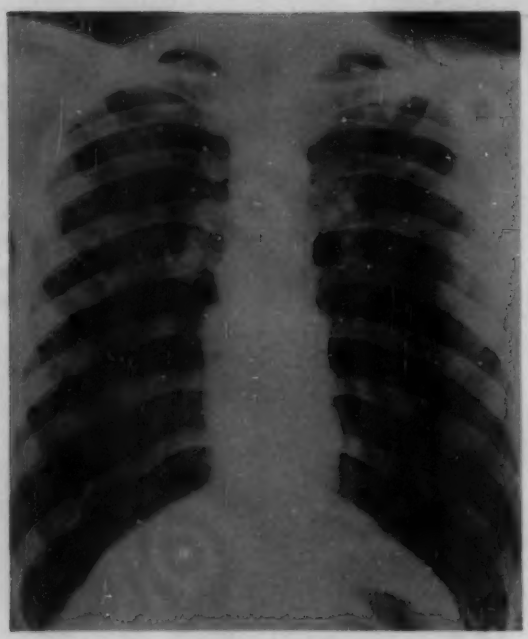


Fig. 11

pressed as percentage variations above or below estimated normal area. The greatest transverse diameter, also suitably corrected for distortion, may also be expressed in a like manner with reference to normal prediction tables. For the case in question area and transverse diameter variations as obtained from the teleroentgenogram were found to be +35 per cent and + 22 per cent respectively. Area and transverse diameter determinations from orthodiagraphic tracings are derived in a similar manner except that here correction for distortion is unnecessary. Determined in this fashion in the case in question, area and transverse diameter yielded results of +25 per cent and +15 per cent respectively.

Although three of the methods illustrated have for the same case yielded evidence of some degree of cardiac enlargement, while the fourth method has failed to detect any such change, and although one feels confident that the clinician's diagnosis of hypertensive heart disease with myocardial degeneration is, in the case of this patient, associated with some slight degree of left ventricular enlargement, none of these methods can be said to have established the actual size of this woman's heart. All of the methods presented are subject to technical errors within themselves, for which due allowance must be made. Because of the fact that on the basis of physical examination alone frank or questionable cardiac enlargement is frequently reported when by the use of roentgen methods no such situation is apparent, it is convenient and gratifying for the roentgenologist to have some means at his disposal for expressing an estimate of heart size. It is, furthermore, reassuring to confirm with mathematically expressed measurements casual impressions of increased or decreased cardiac size, though at all times one should remember the fallibility of such measurements.

Estimations of cardiac volume derived mathematically from frontal area, combined frontal and lateral areas, and computed "real section" have been devised and are used by some workers. The most elaborate procedure of this sort is the actual volumetric reconstruction of the living heart in plastic material carried out by an ingenious application of the orthodiagraphic procedure. Such measures, undoubtedly, have their place in experimental problems concerning the behavior of the heart under various circumstances, and it

Fig. 12

is entirely possible that observations so obtained may one day be helpful in connection with exercise tests in evaluating cardiac reserve. They are, however, in general, too elaborate for routine clinical application.

It has already been pointed out that even under normal conditions the exact anatomical position of the human heart is variable in different individuals and that, furthermore, its anatomical relationships are constantly variable for any one individual. Beyond what may be considered the normal range of such variations in position, the human heart may under a variety of circumstances be displaced from its customary bed to a greater or lesser degree. Any situation which modifies the position, volume or consistency of the thoracic structures which surround the heart may modify its position. Elevation of one or both diaphragms as a result of intra-abdominal lesion, pregnancy, phrenic paralysis or congenital malformation, is particularly prone to produce unilateral displacement or marked rotation of the cardiac axis. Distortion of the chest wall, resulting from scoliosis or kyphosis, deformity of the sternum, congenital or postoperative absence of ribs, all are capable of producing profound degrees of cardiac displacement. Within the chest proper, tumor masses, aneurysms, pleural lesions of all sorts, and lesions which modify the volume of various lung segments are particularly prone to result in cardiac displacement. Examples of such displacement by massive pleural effusion, pressure pneumothorax, bronchial

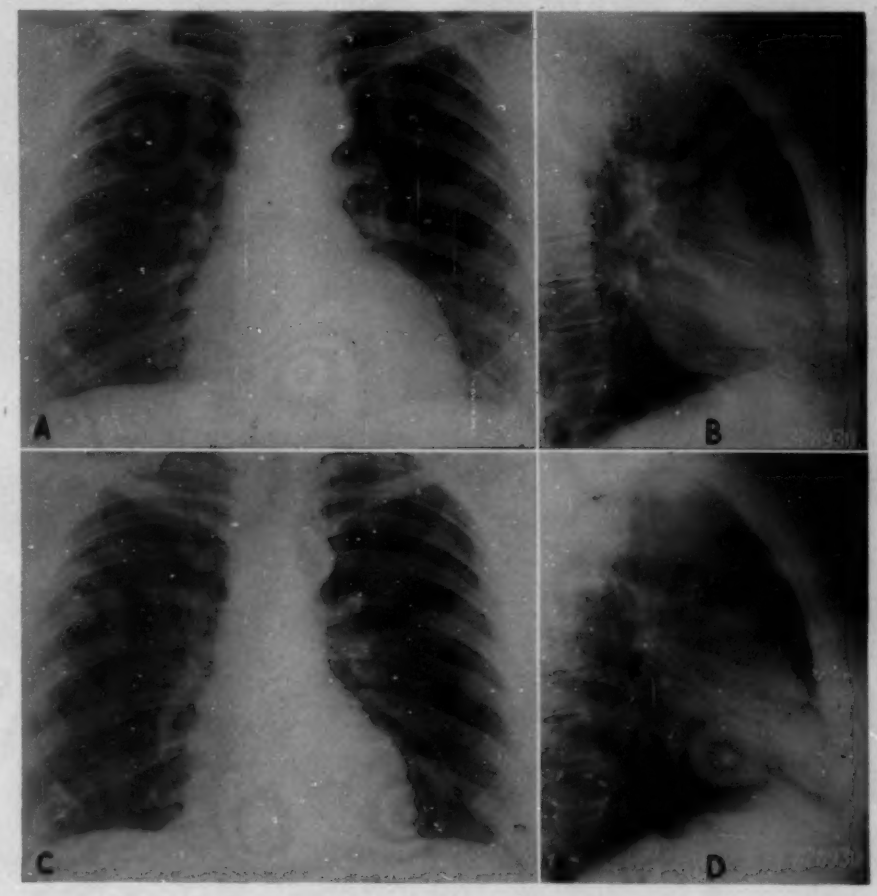


Fig. 13, *A*, *B*, *C* and *D*.

obstruction by foreign body, postoperative massive pulmonary collapse and complete obstruction of a bronchus by neoplasm, are illustrated in Figures 3, 4, 5, 6, and 7, respectively. The unpredictable degrees of rotation which complicate cardiac displacements of this sort render standard projections unreliable for heart size determination by any of the methods in common use. Many of the factors which produce cardiac displacement may at the same time sufficiently obscure the margins of the cardiac silhouette in any or all projections to render impossible any attempt to measure the heart. Any pathological situation which destroys the usual sharp contrast between the heart and surrounding structures will also defeat efforts to estimate cardiac size. Notable among such situations are, of course, pneumonia, extensive pleural thickening, extensive pulmonary fibrosis, far advanced pulmonary tuberculosis, late stages of silicosis, and the condition which is commonly encountered in situations where information regarding cardiac size is of particular importance,

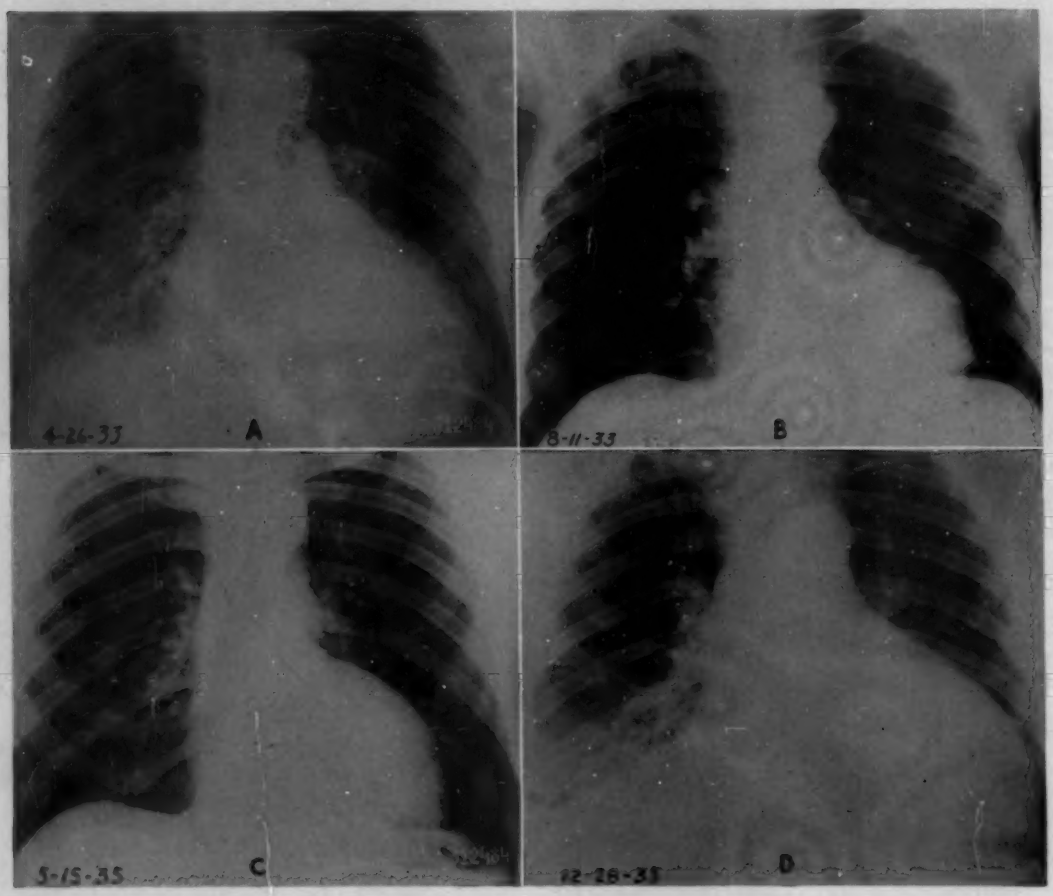


FIG. 14, *A*, *B*, *C* and *D*.

the generalized pulmonary congestion which accompanies cardiac failure. Extreme examples of cardiac contour obliteration produced by enormous generalized broadening of the thoracic aorta in one case, and masses of lymphoblastomatous lymphoid tissue in another are illustrated in Figures 8 and 9, respectively.

Lesions which affect the pericardium are very apt indeed to obscure the shadow of

the heart itself. Figure 10 represents an example of massive pyopericardium before and after surgical drainage.

The wide limits of heart size which one may expect to encounter in examinations of this sort are shown in Figures 11 and 12, the first, an extremely small and narrow cylindrical heart in a patient with high grade emphysema and pulmonary tuberculosis; the second, an enormous heart in a case of long-standing rheumatic heart disease with multiple valvular involvements. Heart size measurements may be really helpful in estimating the extent of progressive change in the size of the heart shadow as shown in Figure 13, representing the heart in the case of hypertensive heart disease before and after the surgical procedure of bilateral splanchnicectomy. In this instance, the frontal plane cardiac area, estimated to exceed normal prediction by 44 per cent, is shown to have decreased to +15 per cent following treatment. In Figure 14, progressive changes in heart size over an approximate two-year period are illustrated in a case of rheumatic heart disease, initially seen in severe decompensation, examined periodically during a period of improvement brought about by thorough digitalization, and finally examined during the second and final episode of cardiac failure. Heart size determinations are of real value in following the course of treatment in cases such as this.

SUMMARY

It is not to be assumed from the foregoing remarks that heart size is unimportant in the diagnosis of cardiac disease; quite the contrary is true. Procedures designed to estimate cardiac size by roentgen methods are available, but it does not follow that measurements of this sort can be made with great exactitude, nor is it true that exact heart size determination is essential to good diagnosis. It is one matter to attempt heart size measurements in selected normal individuals, quite another to attempt such procedures in the case of many individuals where knowledge regarding exact heart size may be of paramount importance to the clinician in determining the diagnosis or observing the effects of treatment. A great variety of situations, commonly encountered by roentgenologists, contrive in one way or another to defeat attempts to provide accurate cardiac measurements.

The continued search by means of experimentation and clinical observation for a truly accurate method of heart size determination during life will and should go on, for if this goal can be achieved new fields of usefulness can be explored. The ambition of cardiologists to develop some reliable method of estimating cardiac reserve and thereby to evaluate with accuracy the disability produced by various cardiac lesions is a worthy one, and perhaps in the future the roentgenologist may be able to help his colleagues in internal medicine in this direction. As matters now stand, it is important that we show good judgment in utilizing the methods at our disposal, that we learn to relegate the matter of heart size to its proper position and importance in cardiac diagnosis.

REFERENCES

Cardiac Diameters and Variations in Shape

1. Danzer, C. S. Cardiothoracic ratio: an index of cardiac enlargement. *Am. J. M. Sc.*, 1919, *157*, 513–521.
2. Dietlen, H. Herz und Gefässe im Röntgenbilde. J. A. Barth, Leipzig.
3. Levene, G., and Reid, W. D. Chart for differential diagnosis of cardiac enlargement by means of the roentgen ray. *Am. Heart J.*, 1932, *7*, 380–382.
4. Vaquez, H., and Bordet, E. The Heart and the Aorta. Translated by James A. Honeij and J. Macy. Yale University Press, New Haven, 1920.
5. Wilson, F. N., and Merrill, E. F. Value of roentgen ray in cardiac diagnosis. *Am. J M. Sc.*, 1923, *165*, 340–356.

Size Compared with Predicted Normal

6. Bainton, J. H. Transverse diameter of the heart. *Am. Heart J.*, 1932, *7*, 331–341.

7. Bardeen, C. R. Determination of size of the heart by means of x-rays. *Am. J. Anat.*, 1918, *23*, 423.
8. Eyster, J. A. E. Size of the heart in the normal and in organic heart disease. *Radiology*, 1927, *8*, 300–306.
9. Eyster, J. A. E. Determination of hypertrophy by roentgen-ray methods. *Arch. Int. Med.*, 1928, *41*, 667–682.
10. Fugmann, W. Zur Beurteilung der Grösse des menschlichen Herzens. Untersuchungen zu dem von H. Rautmann angegebenen Korrelationsverfahren zur Bestimmung der orthodiagraphischen Herzgrösse. *Ztschr. f. d. ges. Anat.* (*Abt. 2*), 1928, *13*, 554–566.
11. Hodges, F. J., and Eyster, J. A. E. Estimation of transverse cardiac diameter in man. *Arch. Int. Med.*, 1926, *37*, 707–714.
12. Hodges, P. C. Comparison of teleoreoentgenogram with orthodiagram. Am. J. Roentgenol. & Rad. Therapy, 1924, *11*, 466–474.
13. Hodges, P. C., Adams, W., and Gordon, W. Estimation of cardiac area in children. *J. Am. M. Ass.*, 1933, *101*, 914–916.
14. Hodges, P. C., and Eyster, J. A. E. Estimation of cardiac area in man. Am. J. Roentgenol. & Rad. Therapy, 1924, *12*, 252–265.

Left Anterior Oblique Diameters

15. Fray, W. W. Mensuration of heart and chest in left posteroanterior oblique position: Comparative study.
I. Relation of the transverse diameter of heart to thorax. Am. J. Roentgenol. & Rad. Therapy, 1932, *27*, 177–186.
II. Determination of type of cardiac enlargement (right or left). *Ibid.*, 363–372.
III. Correlative measurement of aortic arch and the thorax. *Ibid.*, 585–602.
IV. Comparison of the transverse diameters of the heart and the cardiothoracic indices of the chest obtained from the posteroanterior and left anterior oblique chest films. *Ibid.*, 729–739.

Volume Computation

16. Bardeen, C. R. Estimation of cardiac volume by roentgenology. Am. J. Roentgenol., 1922, *9*, 823–832.
17. Hammer, G. Die Herzfläche als Massstab für die Herzgrössenbestimmung. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1928, *38*, 1000–1015.
18. Kahlstorf, A. Ueber eine orthodiagraphische Herzvolumenbestimmung. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1932, *45*, 123–146.
19. Rohrer, F. Volumbestimmung von Körperhöhlen und Organen auf Orthodiagraphischem Wege. *Fortschr. a. d. Geb. d. Rönlgenstrahlen*, 1916–1917, *24*, 285–294.

Volume of Plastic Reconstruction

20. Klason, T. On horizontal orthoprojection of the heart. *Acta radiol.*, 1930, *11*, 57–77.
21. Lysholm, E. Röntgenoskopischer Modellierungsapparat auch für Quersektion und Lokalisation. *Acta radiol.*, 1926, *7*, 189–192.
22. Palmieri, G. G. Ueber meine Methode der plastischen Darstellung des Herzens am Lebenden ("radioplastik"). *Acta radiol.*, 1929, *10*, 127–166.
23. Roesler, H. Clinical Roentgenology of the Cardiovascular System. Charles C. Thomas, Springfield, Ill., 1937.
24. Schatzki, R. Plastische grössen- und lagewahre Darstellung des Herzens. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1928, *37*, 899–905.

DISCUSSION

Dr. George W. Holmes, Boston. Dr. Hodges has presented a very complete and I think conservative discussion of heart measurements. Unfortunately, as he points out, the measurements of the heart, even by the roentgenologic method, are far from accurate, although as all of us would admit, and our clinical friends would I think join us in this admission, it is by far the most accurate method that we have today of measuring the heart or determining its size. When we present figures in fractions of millimeters, it gives the impression that we are doing a very accurate job, but as we all know, we are not. It is really all relative as Dr. Hodges has stated in his paper.

Another thing that I think we should bear in mind is that the importance of determining the size of the heart is constantly becoming less important. Back in 1914, when I first began to do this work, clinicians put much more stress on the size of the heart than they do today. In the cases where they do want our help, in determining whether a given heart shows any increase over the normal, where diagnosis may depend on whether or not enlargement is present, we usually fail. It is very difficult for us to determine in a case that we have never seen before, by comparison with any table which we may have, whether or not that case shows a small amount of enlargement, so that while I think this particular work is of value, we should all know its limitations.

Dr. Samuel Levine, Boston. Heart specialists, I imagine, have needed the roentgenologist less than specialists in any major field of medicine. I have great fear of and great admiration for a roentgenologist because he so often tells me things that I never even suspect, concern-

ing parts of the body—the lungs, the gastro-intestinal tract, the bones, and so forth. But it is not very often that those of us who are interested in clinical heart disease have to lean heavily on the roentgenologist; at least, that has not been so in the past. I suspect it is going to be more so in the future. We have been fairly self-sufficient in our diagnosis, prognosis and treatment with the old-fashioned clinical methods of examination of the sick.

There are some things, however, in which we do need a good deal of help, and one is the very point about which Dr. Hodges spoke—heart size. Any honest clinician will quickly learn that he cannot percuss a normal heart with any high degree of accuracy in many instances. It is just those cases in which we as clinicians are in doubt as to whether or not there is any heart disease, that we need a roentgen examination. It is not the case that is diagnosed readily, but one where I am anxious to know the size of the heart; if I do not know whether or not there is anything wrong with the heart that needs a roentgen examination, because if the roentgen ray shows definitely enlargement of the heart, that heart is diseased. That is the responsibility which the roentgenologist has, and he therefore needs to be cautious in saying that the heart is enlarged.

From what Dr. Hodges has said, we can all see that caution needs to be exercised because of the great variations in the configuration of the heart.

Heart size and heart weight are not the same. A heart can show hypertrophy of 25 per cent and yet have a normal size. The thickness is not measured altogether by the silhouette. We can find that out clinically at times when even the roentgen ray does not help. We can feel the thrust of a left ventricle and know that the ventricle is thickened. There are many instances in which the heart silhouette is definitely enlarged and the roentgenologist can make it out when we clinicians can not. Such information is very valuable to us.

The main point it seems to me to emphasize is that the roentgenologist should be honestly cautious, frank and outspoken when he can say that the heart is definitely enlarged, and if he is not, he should indicate his uncertainty and not try to help a clinician too much.

There are cardiac conditions in which the heart size is normal. There are many patients with angina pectoris—and I mean the lethal type of angina pectoris—who have a normal-sized heart. There are acute rheumatic cardiac conditions, with hearts that are normal in size. There are congenital cardiac conditions with the heart normal in size. In other words, a normal-sized heart does not rule out organic heart disease, whereas a definitely enlarged heart does mean heart disease of some kind.

VISUALIZATION OF THE CHAMBERS OF THE HEART, THE PULMONARY CIRCULATION, AND THE GREAT BLOOD VESSELS IN HEART DISEASE*†‡

PRELIMINARY OBSERVATIONS

By GEORGE P. ROBB, M.D., *and* ISRAEL STEINBERG, M.D.
NEW YORK CITY

THE value of roentgenology in the diagnosis of heart disease has been recognized almost from the discovery of the x-ray by Roentgen in 1895. Shortly thereafter, Holzknecht[26] utilized this method of study and in 1901 reported his observations in the normal and in patients with heart disease. Since then the importance of cardiac roentgenology and the accuracy of interpretation have been established by the pioneer work of Moritz,[34] Dietlen,[10,11] Groedel,[18] Staehelin,[54] Vaquez and Bordet,[61] Bardeen[3] and Assmann,[2] and by the more recent anatomic studies of Smith[63] and Kirch,[27,28,29] the cadaver visualization studies of Laubry and his pupils,[21,31,50] and the clinical investigations of Eyster and his associates,[14,15,24,25] Bedford and Treadgold,[4] Parkinson and his coworkers,[39,41] Nemet,[35] Nemet and Schwedel[36] and Roesler.[49] There have also been significant advances in roentgenologic apparatus and technique which have made possible the clear definition now found in the conventional roentgenogram and fluorescent screen and have also provided valuable new applications of the roentgen ray, such as roentgen kymography,[23,60] cineroentgenography,[44,59] and body section roentgenography[33] which record cardiac and vascular pulsation on the one hand and permit sectional roentgenography of the cardiovascular structures in the thorax on the other.

Despite the obvious importance of roentgenology of the heart, the roentgen ray still gives an incomplete picture of the cardiovascular system, for the four separate chambers of the heart are represented on the conventional roentgenogram and screen by one undifferentiated shadow, and the great blood vessels can be seen indistinctly if at all. As a result, the roentgenologic diagnosis of heart disease has been forced to rely upon indirect evidence such as enlargement of the cardiac shadow,[14,38,39] deformity of one or another segment of the cardiac silhouette,[30,35,55] change in cardiovascular pulsation, and displacement and distortion of the neighboring esophagus,[13,42,45] trachea and bronchi.[50,56,57]

Almost a decade ago, Forssmann[16] recognized the need for separate visualization of the component parts of the heart, and attempted unsuccessfully to accomplish this by the intracardiac injection of the organic iodide compound, uroselectan. Subsequently, the pulmonary blood vessels were visualized by means of Forssmann's method of cardiac catheterization,[7,12,22,43] but the right side of the heart received no attention except for the few observations of Ameuille, *et al.*[1] and the more recent investigation of congenital heart disease in young children by Castellanos and his associates.[5,6] The left cardiac chambers have never been outlined, and the thoracic aorta has been visualized by ventricular and aortic puncture[37] in only two instances.

We have recently developed a practical method for visualizing the four chambers of the heart and the thoracic blood vessels in man and have described it in detail.[46,47] The method consists of two essential parts: (1) the rapid peripheral intravenous injection of enough radiopaque solution (concentrated diodrast—70 per cent) into the

* From the Department of Therapeutics, New York University College of Medicine, and the Third (New York University) Medical Division of Bellevue Hospital.

† This investigation was aided by a grant from the Department of Medical Research of the Winthrop Chemical Company.

‡ Read in a Symposium on Heart Disease at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938.

blood stream to make the interior of the heart opaque to the roentgen ray during the first circulation, and (2) the making of roentgenograms of the cardiovascular structures at the moment of their opacification. The intervals between injection and the arrival of the radiopaque solution in the various parts of the cardiovascular system are remarkably short and should be gauged beforehand by measuring the "arm to lung" and the "arm to carotid sinus" circulation times to the right and the left sides of the heart respectively. By making suitable allowances, the circulation times to intervening points can also be estimated. In the average normal person the superior vena cava, its tributaries, and the right atrium are opacified at one and a half seconds after the beginning of the injection; the right ventricle and the pulmonary arterial tree at three seconds; the pulmonary veins and the left atrium at six to eight seconds, and the left ventricle and thoracic aorta at eight to ten seconds. Because of the location of the heart and the large vessels within the thorax, the position of the patient during visualization should vary with the structures to be outlined; the merits of the various positions will be referred to later. It is important that the optimal degree of rotation for the oblique positions be determined by roentgenoscopy in each case, for the conventional angle of 45° is usually insufficient for the ideal left anterior oblique view and too large for the right oblique position. The roentgenographic technique differs from the conventional only in the use of overpenetration. With one injection and the two roentgenograms provided by the ordinary stereoscopic casette shifter it is possible to visualize the right side of the heart and the pulmonary arterial tree practically every time, and also the left cardiac chambers and the thoracic aorta in about three-quarters of the cases.

Our experience now consists of 263 injections in 140 patients, of whom 44 were normal, 45 had heart disease and 51 pulmonary disease. The immediate reaction to the injection is mild and transient even in patients who are seriously ill; delayed effects are unimportant; and there is no detectable injury to the heart, the lungs, the kidneys, the liver, or the blood. There has been no fatality or other serious consequence. The interpretation of the conventional and the contrast roentgenograms has been based upon the evidence provided by Gray's Anatomy,[17] MacCallum's Pathology,[32] the anatomic and roentgenographic studies referred to previously, as well as those of Schwedel and Epstein,[52] Rubin,[51] and White,[62] and upon our own postmortem study of the heart. Direct confirmation by necropsy has rarely been possible since most of our patients are still alive. The following structures have been visualized in the normal[48] and in heart and lung[58] disease: the superior vena cava and its tributaries; the four chambers of the heart and their walls; the ventricular septum; the pulmonic and aortic valves and their respective sinuses; the entire pulmonary circulation; the thoracic aorta, its wall, and many of its branches and the abdominal aorta. Detailed visualization thus provides an accurate life-size plan of the interior of the heart and the thoracic blood vessels in which each major structure whether normal or diseased can be identified, its characteristics observed, and its size measured.

The purpose of this preliminary report is to illustrate the value of this method of roentgenologic study in the four common types of cardiovascular disease: namely, rheumatic, syphilitic, hypertensive, and arteriosclerotic.

A. RHEUMATIC HEART DISEASE—MITRAL STENOSIS AND INSUFFICIENCY

In mitral stenosis and insufficiency, an increased load is placed upon the left atrium, the pulmonary circulation and the right ventricle by the narrowed mitral orifice, and upon the left ventricle by the regurgitation of blood through the incompetent valve. When the heart fails the right atrium also becomes overworked.

These pathological changes in the circulation lead to dilatation and hypertrophy of the involved chambers, which in turn produce characteristic alterations in the size and shape of the heart. The left atrium which normally lies beside and slightly above the right atrium enlarges in all diameters and in so doing forces its companion to the right, compresses and displaces the yielding esophagus posteriorly and to either side, and may even, it is said, elevate and compress the left main bronchus causing stenosis and an increase in the interbronchial angle. The pulmonary veins, the smaller blood vessels, and the pulmonary artery and its main branches become dilated as a result of the hypertension existing in the lesser circulation. The outflow tract of the right ventricle is elongated upward so that its termination, the conus arteriosus, occupies a higher position than normal and thus accentuates the prominence of the enlarged pulmonary artery. The inflow tract is characteristically widened and its wall thickened, causing transverse enlargement of this cavity. Due to mitral incompetence, the left ventricular wall also becomes hypertrophied and its cavity dilated, causing further enlargement of the transverse axis of the heart. In both ventricles hypertrophy of the wall is accompanied by enlargement of the trabeculae. The aorta is usually normal or hypoplastic, while the right atrium remains normal in size in the absence of congestive heart failure.

The heart lends itself particularly well to roentgenologic study because it lies within the thorax surrounded by the air-containing lungs and consequently it can readily be looked at from many angles. And multiple views are necessary if one is to form an accurate concept of the size and the shape of this irregular three-dimensional structure with its large afferent and efferent vessels. The conventional frontal position gives an oblique view of the heart and the aorta in which the right border is normally formed by the superior vena cava above and by the right atrium below. The left border usually exhibits four curves, the upper one due to the transverse and descending portions of the aorta, the second curve to the pulmonary artery, the next one to the left auricula, and the lowest curve to the left ventricle. In this view the diaphragmatic contour is formed by the right and left ventricles, the former contributing approximately three-fifths of it.

The usual right anterior oblique projection gives a side view of the heart in which the two atria form the posterior border, the left atrium lying above and remotely and the right one below and nearer to the film. The ill-defined diaphragmatic contour of the heart in this projection is formed by the inflow tract of the right ventricle; while the apical portion of the silhouette is produced by the left ventricle and the pulmonary arc by the pulmonary artery and possibly by the conus arteriosus[35,41] in small part. The receding left branch of the pulmonary artery is frequently seen as an oval density marking the bifurcation of this vessel, while the right branch can at times be seen crossing the spine. The aorta is poorly defined. An end view of the heart is obtained in the conventional left oblique position in which the right and the left chambers lie side by side separated by the ventricular septum, and the aorta crosses to the left in side view. The right contour is formed by the right auricula above and the inflow tract of the right ventricle below; and, similarly, the left border is formed by the left atrium and ventricle. The aortic arch encloses an open space, the so-called "aortic window," in which the left branch of the pulmonary artery is frequently visible. The trachea and the two main bronchi can be seen to advantage in this position, the left bronchus especially well as it passes obliquely downward above the left atrium.

In *mitral stenosis and insufficiency* the pathologic changes cause characteristic alterations in the size and shape of the heart which are reflected in the cardiac silhouette. In the frontal view (Fig. 1) the heart is enlarged in all diameters and is triangular in shape due to the prominence of the right atrial and the left ventricular con-

tours below and the convexity of the pulmonary arch above. The right cardiac border exhibits the double contour frequently seen in this condition of which the upper curve is produced by the enlarged left atrium and the lower one by the right atrium. In this case the left border consists of four curves: the upper one, the aortic, is more prominent than that usually found in this disease; the accentuated second and third curves are formed by the dilated pulmonary artery and the enlarged left auricula respectively, although the pulmonary conus has been ascribed a prominent rôle in the enlargement of the pulmonary arc by some observers. The prominent lower border is formed by the enlarged and displaced left ventricle.

The characteristic changes in the right oblique position are the enlargement and triangular shape of the heart, the obliteration of the retrocardiac space, the displacement and stenosis of the esophagus by the enlarged left atrium, and the prominence of the pulmonary arc due to enlargement of the pulmonary artery and lengthening of the outflow tract. In the left oblique view, the enlargement of the left atrium causes prominence of the upper left cardiac border and may, it is said, be responsible for the elevation and stenosis of the left bronchus. Enlargement of the right and and left ventricles is indicated by the increased width of the heart toward the right and left sides respectively and by the greater convexity of the lower cardiac borders. The inflow tract of the right ventricle alone causes the prominence of the lower right border.

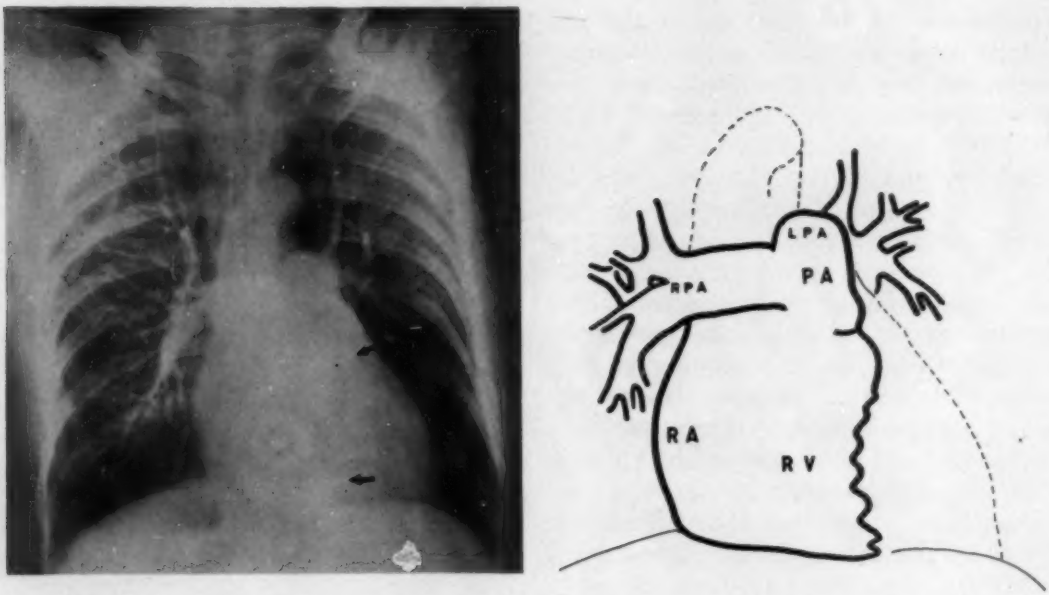
It is apparent from this brief review that much can be learned regarding the pathology of the heart in mitral stenosis and insufficiency by conventional roentgenology but that precise information concerning the nature and extent of the abnormalities in the individual case cannot be obtained. Such data, however, are provided by our method of contrast roentgenography of these structures.

J. H., aged forty-eight. Conventional roentgenogram (Fig. 1). The cardiac silhouette was generally enlarged, showing prominence of both lower borders and the pulmonary arc. The right side exhibited a double contour. On the left side the left ventricular segment was unduly prominent as was the middle arc with its two eminences. The ascending aorta was small but the aortic knob appeared prominent. The pulmonary blood vessels were enlarged throughout

Fig. 1. J. H., aged forty-eight. Rheumatic heart disease, mitral stenosis, and insufficiency. Conventional roentgenogram. Note enlargement and characteristic configuration of cardiac silhouette, double contour on right side of heart, two prominent convexities in middle region of left side, and increased lung markings.

but the lung fields were clear. The cardiac silhouette was characteristic of rheumatic mitral stenosis and insufficiency.

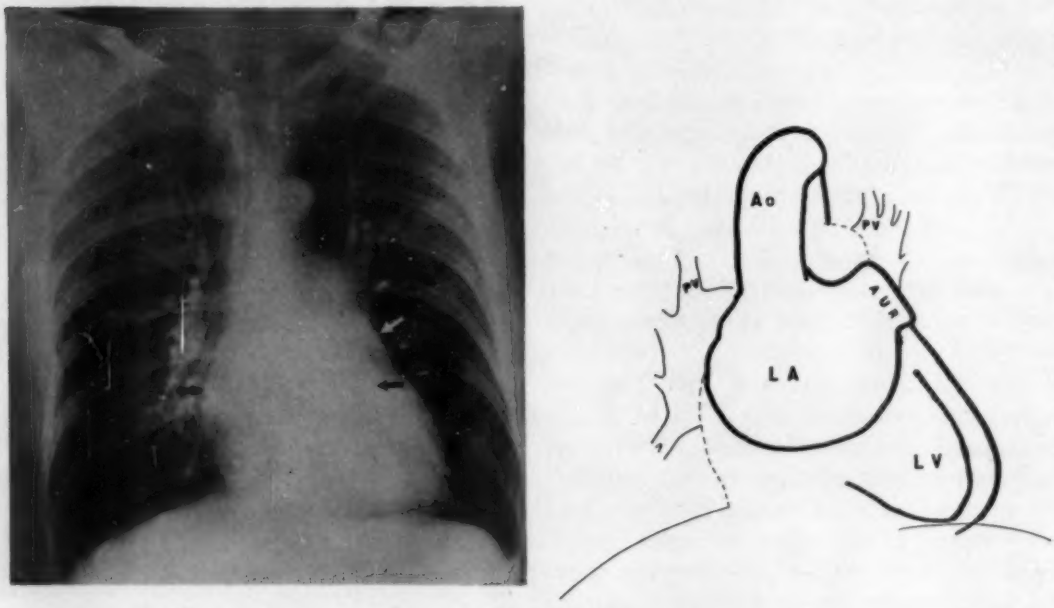
Contrast roentgenogram (Fig. 2 *A* and *B*); frontal position at four and a half seconds after beginning of injection: The right atrium and ventricle, the pulmonary artery with its right and left branches and their subdivisions were opaque, but the superior vena cava was no longer outlined. The right atrium was well filled but could not be differentiated from the right ventricle which lay in front of and obscured its left border. The ventricular septum extended well beyond the left border of the spine and its border was irregular and concave toward the left. In this view the right side of the heart comprised two-thirds of the diaphragmatic contour, the remainder being formed by the left ventricle. Only the left border of



A *B*

FIG. 2. *A*. J. H. Rheumatic heart disease, mitral stenosis and insufficiency. Contrast roentgenogram at four and a half seconds after start of injection. Right atrium with right ventricle and pulmonary arterial tree opaque. Lower arrow denotes ventricular septum—upper arrow conus arteriosus and pulmonary valve. Note conus is not border forming; also enlargement and greater number of pulmonary arterial vessels.

B. Tracing of *A*. RA = right atrium; RV = right ventricle; PA = pulmonary artery; RPA = right pulmonary artery; LPA = left pulmonary artery. Note left cusp of pulmonary valve.

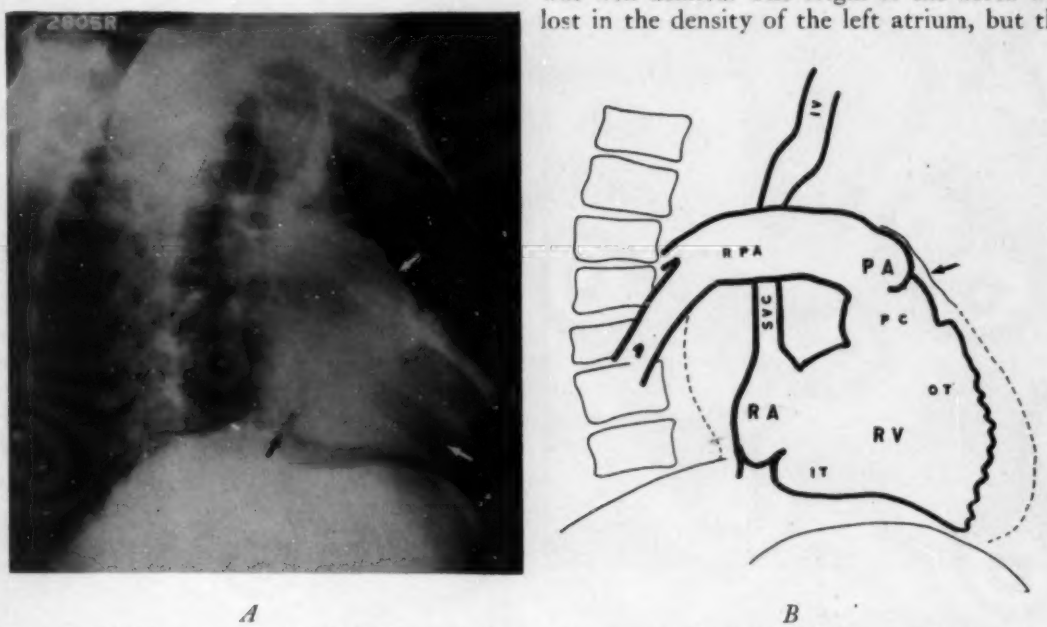


A *B*

FIG. 3. *A*. J. H. Rheumatic heart disease, mitral stenosis and insufficiency. Contrast roentgenogram at eighteen seconds. Pulmonary veins, left atrium, left ventricular cavity and wall, and aorta outlined. Body of left atrium bracketed by two lower arrows; note enlargement to right and failure to reach left border. Upper arrow denotes left auricula. Note increase in size and number of pulmonary veins.

B. Tracing of *A*. PV = pulmonary veins; LA = left atrium; LV = left ventricle; AO = aorta; AUR = left auricula.

the pulmonary conus was visible and it was not border forming but lay 1.4 cm. within the cardiac mass. The left pulmonary cusp was well defined, thus indicating the beginning of the pulmonary artery. The pulmonary artery appeared enlarged as did its right branch which measured 3 cm. in diameter as it crossed the spine horizontally; its inferior branch measured 2 cm. at the hilum. The shadow of the receding left branch was superimposed on that of the pulmonary artery like a cap and could not be differentiated from it except at the superior pole. The entire pulmonary arterial tree seemed to be enlarged.



A *B*

FIG. 4. *A*. M. H., aged thirty-eight. Rheumatic heart disease, mitral stenosis and insufficiency. aortic insufficiency. Contrast roentgenogram—right anterior oblique position at four seconds. Innominate vein, superior vena cava, right atrium, right ventricle, pulmonary artery and left branch filled. Black arrow indicates coronary sulcus separating right atrium and ventricle. Inflow tract extends from tricuspid orifice to apex of right ventricle delimited by lower white arrow. Outflow tract extends from apex to pulmonary orifice indicated by pulmonary cusps and upper white arrow. Note widening of conus arteriosus and pulmonary artery and branches.

B. Tracing of *A*. IV = innominate vein; SVC = superior vena cava; RA = right atrium; RV = right ventricle; IT = inflow tract; OT = outflow tract; PC = pulmonary conus; PA = pulmonary artery; RPA = right pulmonary artery. Arrow points to pulmonary valve.

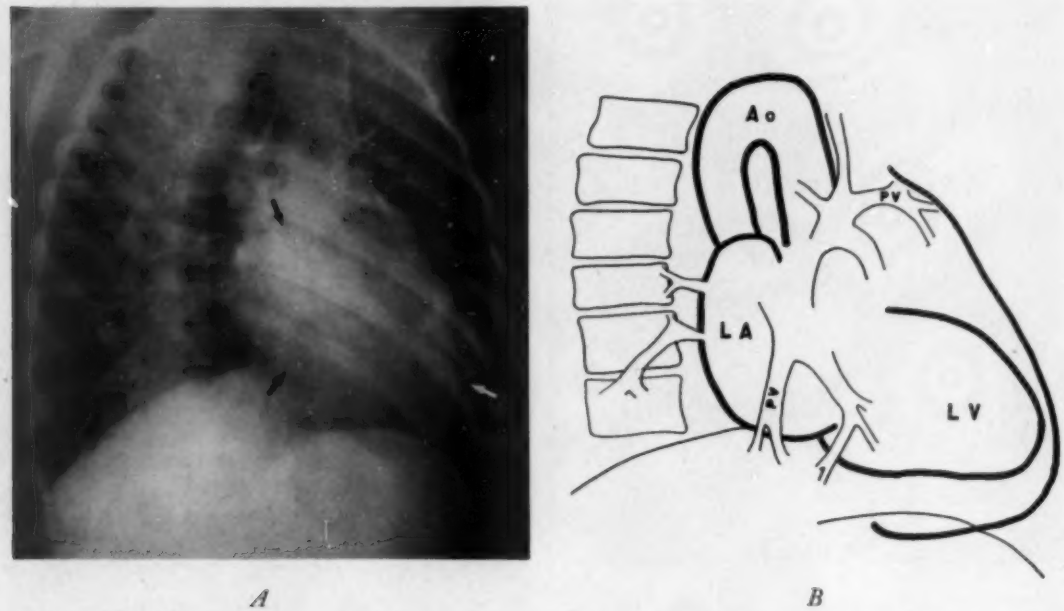
Contrast roentgenogram (Fig. 3 *A* and *B*); frontal position at eighteen seconds: The pulmonary veins, the left atrium including the auricula, the left ventricular cavity, and the thoracic aorta were visualized. The pulmonary veins appeared abnormally prominent as they converged toward the hilum, but particularly so on the left side where a large group united. The left auricula, a narrow projection, formed the lower eminence of the middle contour, while the tremendously enlarged main body of the left atrium extended from the right border of the heart to within a centimeter of the left middle contour. The horizontal diameter of the atrium was 10.4 cm. and the vertical diameter 8.2 cm. The wall of the left ventricle appeared to be moderately thickened, measuring 1.4 cm., and the ventricular cavity was somewhat enlarged. The ventricular septum was well defined. The origin of the aorta was lost in the density of the left atrium, but the upper part of the ascending aorta, measuring 2.6 cm., and the transverse aorta could be seen distinctly. The thickness of the aortic wall at the knob was 2 mm. The origin of the innominate, the left common carotid and the subclavian arteries at the arch was visible and these vessels could be followed to the neck and the arms.

M. H.,* aged thirty-eight. Contrast roent-

* This patient had aortic insufficiency in addition to a double mitral lesion. His roentgenograms are included because of the excellent visualization of the right ventricle and the left atrium in the right oblique projection.

genogram (Fig. 4 *A* and *B*); right anterior oblique position at four seconds: The innominate vein, the superior vena cava, the right atrium, the right ventricle, the pulmonary conus, and the pulmonary artery with its two branches were outlined. The right atrium, measuring 5.5 cm. in vertical diameter and 2.7 cm. horizontally, rested upon the diaphragm and was separated from the right ventricle by the coronary sulcus. The inflow tract of the right ventricle was widened and elongated, its long diameter measuring 9 cm. The outflow tract also was lengthened (10.5 cm.) and its upper end, the pulmonary conus, was enlarged transversely to a diameter of 4.2 cm. The right ventricle extended almost to the apex of the heart and ended as a jagged line. The left pulmonary cusp was clearly outlined, indicating with certainty the end of the conus arteriosus and the beginning of the pulmonary artery. This vessel and its branches were dilated, the main stem measuring 3.4 cm. and the right trunk 2.2 cm. in diameter.

Contrast roentgenogram (Fig. 5 *A* and *B*); right anterior oblique position at ten seconds: The pulmonary veins and the left atrium were clearly visualized, whereas the left ventricle and the aorta were only faintly outlined. The strikingly enlarged left atrium measured 8.0 cm. in vertical diameter, but the horizontal diameter could not be determined accurately because the anterior boundary could not be established. The enlarged right pulmonary veins could be seen crossing the spine and the retrocardiac space.



A *B*

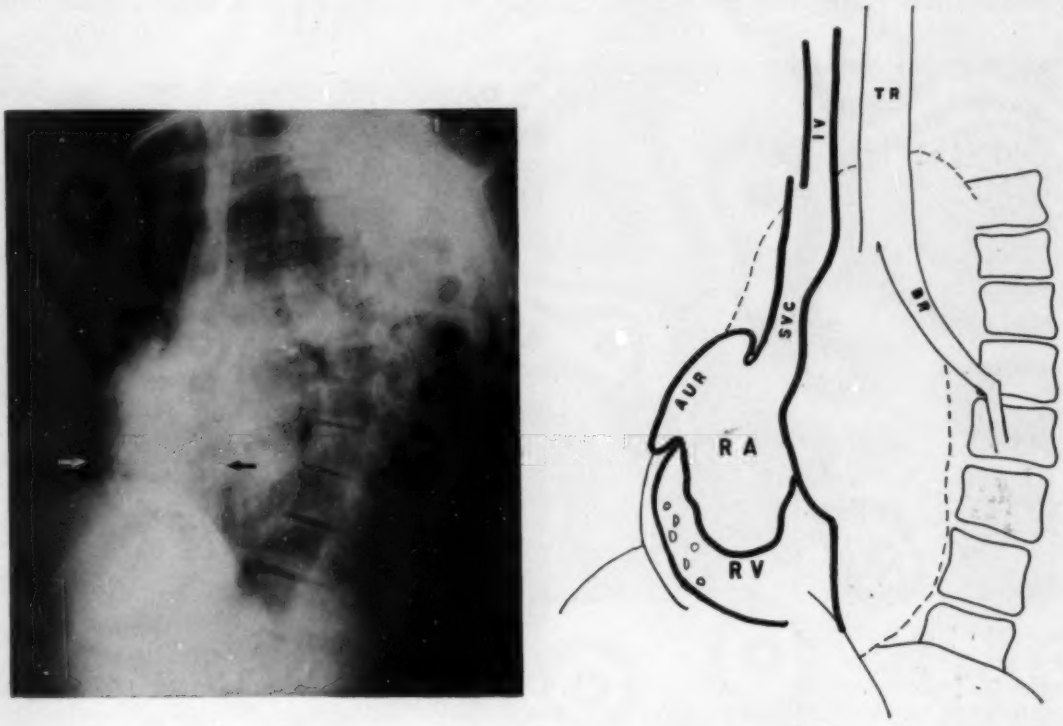
FIG. 5. *A*. M. H. Rheumatic heart disease, mitral stenosis and insufficiency; aortic insufficiency. Contrast roentgenogram—right anterior oblique position at ten seconds. Enlarged pulmonary veins and left atrium well filled. Left ventricle and aorta dimly outlined. Upper black arrow denotes upper pole of left atrium and lower black arrow, coronary sulcus. White arrow lies on left ventricular wall pointing to cavity. Thoracic aorta arches above upper black arrow.

B. Tracing of *A*. PV = pulmonary veins; LA = left atrium; LV = left ventricle; AO = aorta. Note enlargement of pulmonary veins.

M. T., aged seventeen. Contrast roentgenogram (Fig. 6 *A* and *B*); left anterior oblique position at three seconds: The innominate vein, the right atrium, the right auricula, and the inflow portion of the right ventricle were filled. The right innominate vein passed downward to enter the superior vena cava immediately above the aortic arch; and the superior vena cava, exhibiting a shallow groove on its posterior surface at the level of the right pulmonary artery, terminated in the right atrium which was seen as an oval density lying behind the main mass of the right half of the heart. Above and to the right of the atrium was the well-filled ear-like appendage called the auricula.

The larger, less opaque inflow tract of the right ventricle lay in front of the main body of the atrium; its medial border was angular while the outer wall was rounded. The ventricular wall measured 7 mm. and the auricular wall 2 mm. in thickness. The honeycombed appearance of the inflow tract was probably due to the hypertrophied trabeculae carnae. The dilated pulmonary artery which practically filled the entire window showed only slight opacification.

third of the right cardiac border and at this point was 1 mm. in thickness. The medial border of the inflow tract of the right ventricle was obscured by the density of the right atrium but the lateral border was distinctly outlined and its wall measured 4 mm. The outflow tract could be identified only at the upper end where the inner border of the conus arteriosus was visible, the outer margin being covered by the right auricula. Immediately above the conus, two cusps of the pulmonary



A *B*

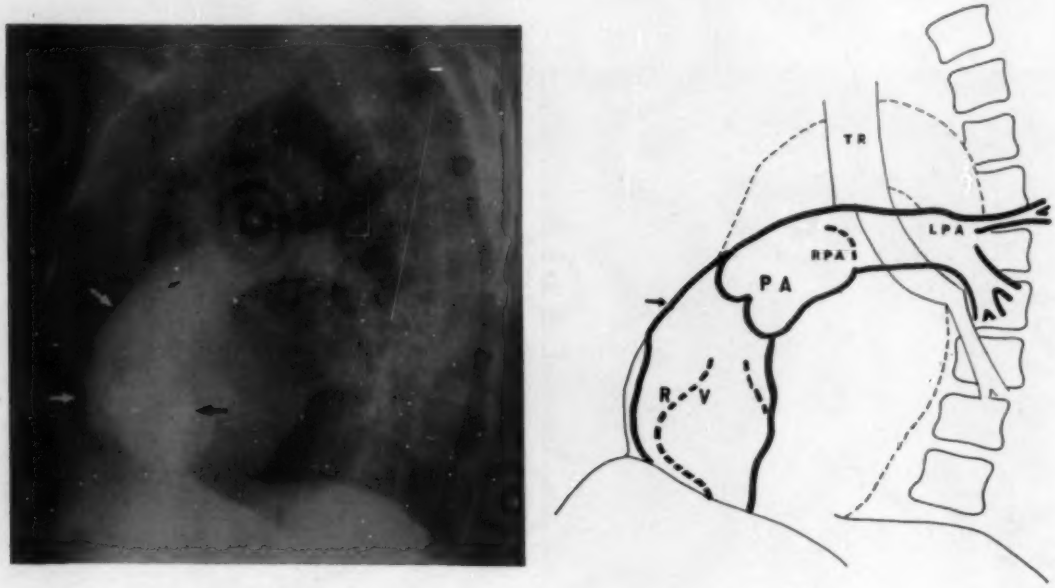
Fig. 6. *A*. M. T., aged seventeen. Rheumatic heart disease, mitral stenosis and insufficiency. Contrast roentgenogram—left anterior oblique position at three seconds. Right innominate vein, superior vena cava, right atrium, right auricula and inflow tract of right ventricle opacified. Arrows bracket inflow tract of right ventricle lying below and in front of atrium. White arrow points to outer surface of ventricular wall and toward trabeculations. Note elevation and stenosis of left main bronchus and impression on superior vena cava by right pulmonary artery.

B. Tracing of *A*. IV = innominate vein; SVC = superior vena cava; AUR = right auricula; RA = right atrium; RV = right ventricle; TR = trachea; BR = left bronchus.

J. H. Contrast roentgenogram (Fig. 7 *A* and *B*); left anterior oblique position at four seconds: The right atrium, the right auricula, the right ventricle, and the pulmonary artery with its branches and subdivisions were filled. The right atrium lying immediately above the diaphragm was seen as a round, dense opacity measuring 5.1 cm. in horizontal diameter. Its auricula extended upward to form the upper valve with their corresponding sinuses, and the dilated pulmonary artery which measured 4.2 cm. in diameter were outlined. The receding right branch of this vessel appeared as an oval density having a diameter of 3.0 cm., while the left branch which crossed beneath the aorta also measured 3.0 cm. at the bifurcation. The subdivisions of both branches of the pulmonary artery were enlarged.

J. H. Contrast roentgenogram (Fig. 8 *A* and *B*); left anterior oblique position at eighteen and a half seconds: The pulmonary veins, the left atrium, and the thoracic aorta were opacified. The pulmonary veins could be seen entering into the left atrium which occupied the superior posterior part of the vertebral border of the heart. The atrium measured 8.4 cm. in vertical diameter and 6.7 cm. in the horizontal. The elevated left main bronchus lay well above and separated from the body of the left atrium and seemed to rest upon an enlarged, rounded mass which was not definitely identified but probably consisted of a group of large pulmonary veins. The left ventricle was poorly filled—probably because it was in contraction. The outer border of the thoracic aorta including one sinus was clearly defined, but the aortic cusps and the medial border of the ascending aorta near its origin were invisible. The aorta measured 2.7 cm. in diameter immediately beyond the origin of the left subclavian artery.

Comment. The size, the shape, and the location of the enlarged left atrium and its relationships with the rest of the heart and the adjacent structures are shown in Figures 3, 5, and 8. This chamber is composed of two parts, a main rounded body which will be referred to as the left atrium in this paper, and an ear-like appendage, the auricula. In the frontal view (Fig. 3) the enlarged left atrium extends from the right border of the heart to within a centimeter of the left border and the auricula forms the convexity immediately below the pulmonary arc. In the right oblique position (Fig. 5) the oval-shaped left atrium encroaches upon the retrocardiac space and towers above the normal-sized right atrium (Fig. 4). The great transverse enlargement of the left atrium is best shown in the left oblique position (Fig. 8). This view also demonstrates that the left atrium, at least in this case, is not directly responsible for the elevation and stenosis of the left bronchus.

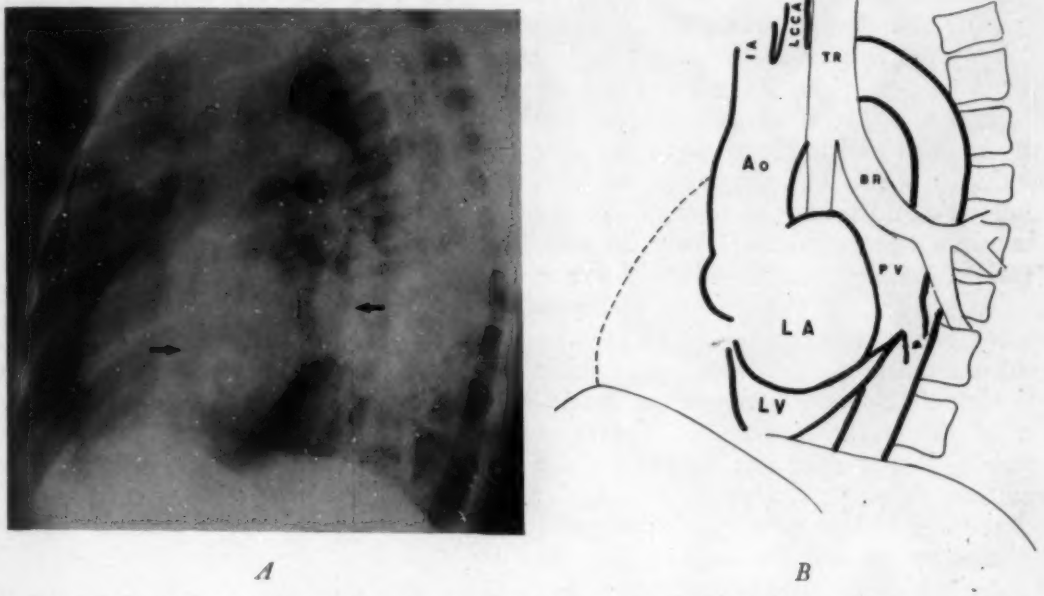


A *B*

FIG. 7. *A*. J. H. Rheumatic heart disease, mitral stenosis and insufficiency. Contrast roentgenogram—left anterior oblique position at four seconds. Right atrium, auricula and ventricle, pulmonary artery and branches opacified. Medial black arrow points to ventricular septum; lower white arrow indicates inflow tract with wall of right ventricle. Upper white arrow denotes auricula. Small black arrow lies on pulmonary artery pointing into cusp. Note right and left pulmonary arteries.

B. Tracing of *A*. RV = right ventricle; PA = pulmonary artery; RPA = right pulmonary artery; LPA = left pulmonary artery; TR = trachea; Note right atrium indicated by broken line. Arrow points to right auricula.

The right ventricle is roughly triangular in shape, consisting of a horizontal inflow tract and a vertical outflow tract (Fig. 4). There is obvious elongation of the outflow tract, causing elevation of the conus arteriosus and the pulmonary artery, both of which form the pulmonary arc in the right oblique position. There is also widening of the conus. The inflow tract is enlarged vertically (Fig. 4) and transversely and its wall is thickened (Figs. 6 and 7). In the frontal view the enlarged right ventricle reaches the surface of the heart only at the diaphragm where it forms a predominant part of this contour, but it does not enter directly into the formation of either the lower right border or the pulmonary arc. The main stem of the pulmonary artery and the right and left main branches appear dilated but not lengthened and their walls are not appreciably thickened. In some instances, the enlarged right pulmonary artery may even compress and deform the superior vena cava lying anterior to it (Fig. 6). There is a striking increase in the size and the number of the smaller arterial and venous blood vessels throughout the lungs and in the size and the prominence of the larger pulmonary veins (Figs. 3 and 5). In fact it seems probable from the observations made in this case and others not included in this report that the engorged left pulmonary veins and not the dilated left atrium are responsible for the compression and displacement of the left bronchus in mitral stenosis.



A *B*

Fig. 8. *A*. J. H. Rheumatic heart disease, mitral stenosis and insufficiency. Contrast roentgenogram—left anterior oblique position at eighteen and a half seconds. The pulmonary veins, left atrium, and thoracic aorta filled. Posterior arrow points to enlarged pulmonary veins, anterior arrow to right border of left atrium. Note enlarged veins and elevated left bronchus.

B. Tracing of *A*. PV = left pulmonary veins; LA = left atrium; LV = left ventricle; Ao = aorta; IA = innominate artery, LCCA = left common carotid artery; TR = trachea; BR = left bronchus.

Good visualization of the left atrium, the left ventricle, and the aorta has been particularly difficult in mitral disease because of the stasis and the increased amount of blood in the pulmonary circulation and the left atrium which cause a marked degree of stringing out of the opaque material in its passage to the left ventricle. However, it has been possible to show that the left ventricular cavity is

definitely dilated and the wall thickened (Figs. 3 and 5). The right atrium and the aorta are not remarkable.

In summary, detailed structural visualization of the heart in our small series of 6 patients with rheumatic mitral stenosis and insufficiency has demonstrated the following points during life: (1) the left atrium enlarges toward the right side in the frontal view, forming the upper curve of the double contour, but it does not reach the left side except as the auricula which forms the eminence below the pulmonary arc; (2) the elevation and stenosis of the left bronchus appear to be caused by the enlarged left pulmonary veins and not the left atrium; (3) the accentuation of the hilar shadows and the increase in the lung markings are due to enlargement of both the arterial and venous components of the pulmonary circulation; (4) the main stem of the pulmonary artery is dilated but not lengthened, and it alone forms the pulmonary arc in the frontal view; (5) the conus arteriosus is dilated and lies farther to the left than normally due to the elongation of the outflow tract of the right ventricle, but it does not enter into the formation of the pulmonary arc in the frontal view and plays only a minor rôle in the right oblique projection; (6) the enlargement of the right ventricle is due to dilatation and hypertrophy of the broad horizontal inflow tract and elongation of the narrow vertical outflow tract with some widening of the conus arteriosus. In frontal view this chamber is broadened, forming three-fifths of the diaphragmatic contour; (7) the left ventricular cavity is moderately dilated and the wall thickened; (8) the superior vena cava, the right atrium, and the aorta remain normal in the absence of heart failure.

B. SYPHILITIC CARDIOVASCULAR DISEASE—AORTIC ANEURYSMS

In the cardiovascular system, syphilis characteristically produces destruction of the media of the arteries, which leads to weakening and dilatation of the wall and thickening of the intima. In the aorta the process involves the ascending portion most frequently, although both the arch and descending portions may be affected. The resulting dilatation is usually generalized but may be localized and either fusiform or saccular in shape. In the absence of aortic insufficiency, ostial stenosis of the coronary arteries and direct involvement of the myocardium by diffuse inflammatory reaction or gumma formation, the cardiac muscle remains unaffected.

The clinical recognition of syphilitic aortitis offers unusual difficulties because there are no pathognomonic signs or symptoms early in the disease and roentgenologic examination gives no help, because the intracardiac portion of the aorta, a common site for the disease, is hidden in the cardiac shadow. When the process is more advanced the roentgen ray will frequently reveal the presence of aortic disease, although adjacent densities may interfere; but, in any event, the exact size and shape of the diseased area cannot be visualized.[19,20] As previously described, satisfactory roentgenologic examination requires study of the heart and aorta in the three conventional views. In the frontal position (Fig. 9) dilatation of the aorta is indicated by the prominence of the supracardiac shadow on the right side which extends well beyond the right atrial border of the heart, and by the elevation and prominence of the aortic knob and descending aorta. The bulging of the ascending aorta suggests aneurysmal dilatation but since the left border of this portion cannot be seen, it remains only a probability. In view of the calcified plaque in the aortic knob it is conceivable that the widening of the aortic loop is due to the unfolding of an arteriosclerotic aorta rather than to syphilitic dilatation.

In the left oblique roentgenogram, not included in this study, there was no unfolding of the aorta, but there was quite marked prominence of the outer border of the ascending aorta, while the inner wall was completely obscured by other densi-

ties. The descending aorta showed a small protuberance on its inner surface immediately below the arch which was not well outlined. In the right oblique view the ascending aorta appeared widened but poorly defined throughout and the descending aorta was not visible. In all positions the heart itself appeared to be normal. Conventional roentgenography thus indicated the presence of a fusiform and a saccular aneurysm in this advanced case of syphilitic aortitis, but it failed to outline these structures or to tell their size.

W. M., aged forty-nine. Conventional roentgenogram (Fig. 9); frontal position: The heart was normal in size and shape, but the supracardiac shadow was widened and measured 10.1 cm. The ascending aorta was enlarged to the right, projecting well beyond the right cardiac border below. The aortic knob was elevated and enlarged to the left and showed a line of calcification 4 mm. from its outer border. From this point the less dense descending aorta curved downward to overlie the spine at the diaphragm. The lung fields were clear and there was an azygos fissure. There was a rounded density at the left hilum probably due to the left pulmonary artery.

Fig. 9. W. M., aged forty-nine. Syphilitic cardiovascular disease, aortic aneurysms. Conventional roentgenogram—frontal position. Note normal-sized heart as contrasted to widened supracardiac shadow. Arrow against outer wall of aortic knob; inner border defined by calcified plaque.

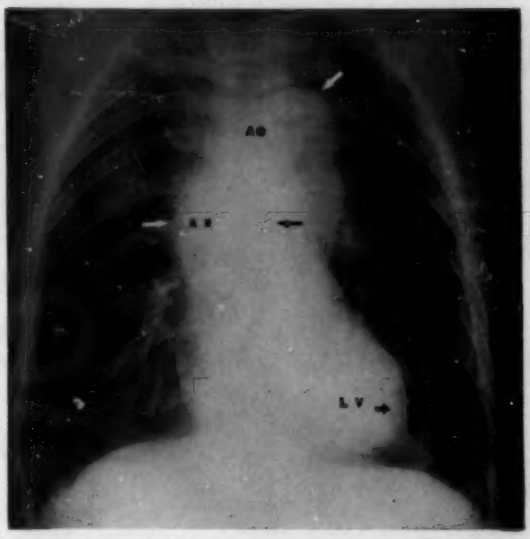


Fig. 10. W. M. Syphilitic cardiovascular disease, aortic aneurysms. Contrast roentgenogram—frontal position at fifteen seconds. Pulmonary veins, left ventricle and aorta opaque. LV = left ventricle; AN = aneurysm of ascending aorta. Lowest arrow indicates inner border of left ventricular wall. Aneurysm bracketed by arrows. Highest arrow against outer border of aortic wall; inner border defined by opaque lumen and calcified plaque. Note displacement of superior vena cava to right.

Contrast roentgenogram (Fig. 10); frontal position at fifteen seconds: The pulmonary veins, the left ventricular cavity and the aortic arch were opaque. The left ventricular cavity was normal in size and shape and the wall measured 1 cm. The origin of the aorta was obscure, but the remainder of the ascending portion and the transverse aorta were clearly defined. The fusiform dilatation of the ascending aorta was 6.3 cm. in diameter and caused the marked projection toward the right. At this point the aorta and superior vena cava together formed the right supracardiac border. Above this level the right border was formed by the superior vena cava while the aorta curved to the left across the spine and measured 4.2 cm. in diameter immediately before the knob. The opaque interior of the aorta at the knob extended to the line of calcification and the aortic wall at this point measured 3.5 mm. Only the outer border of the descending aorta could be seen and it was smooth in contour.

Contrast roentgenogram (Fig. 11); left anterior oblique position at fifteen seconds: The left atrium and the entire thoracic aorta were

FIG. 11. W. M. Syphilitic cardiovascular disease, aortic aneurysms. Contrast roentgenogram—left anterior oblique position at fifteen seconds. AN = aneurysm; LA = left atrium. Left atrium and thoracic aorta opaque. Note dilated aortic sinuses indicated by lowest arrow, fusiform aneurysm of ascending aorta bracketed by arrows, and saccular aneurysm of descending aorta.

opaque, but the left ventricle was not outlined. Two aortic sinuses and cusps were well visualized. Immediately above them the dilated aorta could be seen and measured 4.6 cm. in diameter, whereas the mid-portion of the ascending aorta, the site of maximal dilatation, measured 6.4 cm. It then decreased in size until its diameter was 3.5 cm. beyond the subclavian artery. There was also a saccular aneurysmal dilatation which appeared as a rounded knob immediately below the aortic arch, the aorta at this point measuring 5.7 cm. in width. The width of the aorta at the diaphragm was 2.7 cm.

Contrast roentgenogram (Fig. 12); right anterior oblique position at fourteen seconds: The left ventricle and thoracic aorta were visualized. The left ventricle was poorly defined, but the origin of the aorta including the aortic cusps and sinuses was well outlined. The ascending aorta was dilated fusiformly and immediately above the sinuses at its narrowest point it measured 4.6 cm. in diameter, while the maximal transverse diameter of the aneurysm was 6.4 cm. The aortic wall was thickened and measured 3.5 mm. The aneurysmal bulge of the descending aorta was not visualized.

Comment. The advantage of visualization is illustrated by Figures 10, 11, and 12. As had been shown by conventional roentgenology, the heart was not enlarged and the visualized left ventricle and wall were normal. The intracardiac portion of the aorta (Figs. 11 and 12) was clearly outlined, revealing the essential features of this important but heretofore invisible portion. The dilatation involving the entire circumference of the aorta began at the level of the aortic sinuses (Valsalva) and reached its maximum just before the beginning of the arch. Thereafter the aorta became narrowed to approximately the normal size at the mid-portion of the arch but soon became dilated again to form a saccular aneurysm on the inner border of the descending aorta (Fig. 11). The concentric nature of the aneurysm of the

FIG. 12. W. M. Syphilitic cardiovascular disease, aortic aneurysms. Contrast roentgenogram—right anterior oblique position at fourteen seconds. AO = aneurysm; LV = left ventricle. Left ventricle and ascending aorta filled. Note aortic sinuses and cusps, indicated by lower arrow. Upper arrow denotes aortic wall.

ascending aorta is proved by the similar appearance and identical measurements in the frontal and two oblique positions. On the other hand, the saccular shape of the second aneurysm is demonstrated by its presence only in the left oblique roentgenogram (Fig. 11) in which it is seen in profile and its absence in the other two projections. The aortic wall was moderately thickened as shown by the calcified plaque in the intima at the aortic knob and the opacification of the lumen. Moreover, the exact agreement between the outer limit of opacification and the line of intimal calcification provides strong support for our belief that the shadow in the contrast roentgenogram attributed to the aortic wall actually represents this structure. The remainder of the descending aorta was normal. The superior vena cava was displaced to the right apparently by the aneurysm since its right border coincides with that of the fusiform dilatation which lies far to the right of its normal position.

In summary, visualization of the heart and the aorta in this case of syphilitic heart disease excluded gross anatomical change in the heart, and permitted the demonstration of disease of the intracardiac portion of the aorta which has previously been impossible during life. It thus made possible the determination of the exact size, shape, and location of the aneurysms and the thickness of the aortic wall.

C. HYPERTENSIVE HEART DISEASE

Arterial hypertension places an increased load upon the cardiovascular system resulting in hypertrophy and dilatation of the left ventricle and dilatation and unfolding of the aorta. In long standing hypertension the left atrium may also be dilated and hypertrophied in the absence of detectable left ventricular failure.[39] When left ventricular failure develops, it produces engorgement of the pulmonary circulation, dilatation, and hypertrophy of the right ventricle and finally, with the occurrence of right ventricular failure, dilatation of the right atrium and the superior vena cava.

In hypertensive as in other types of heart disease, the gross pathologic changes of the cardiovascular system are shown by the conventional methods of roentgenologic examination. In the frontal view enlargement of the left ventricle produces outward displacement and characteristic rounding of the left cardiac border immediately above the apex and thickening of the "waist" of the heart so that the cardiac silhouette becomes oval simulating that found in aortic valvular disease. In this view the dilatation of the aorta is demonstrated by a slight lowering of the cardiovascular junction on the right, prominence and displacement of the aortic knob to the left, and convexity and increased density of the descending aorta (Fig. 13). These changes are manifested in the left oblique projection by enlargement and greater convexity of the left ventricular border, dilatation of the thoracic aorta and rounding of the aortic loop often referred to as "uncoiling"[8,9] or "unfolding" of the aorta (Fig. 15). The involvement of the other chambers and the pulmonary circulation as a result of circulatory failure produces further alterations in the roentgenologic picture but these will not be described in this report.

A. W., aged fifty-five; blood pressure 245/145. Conventional roentgenogram (Fig. 13); frontal position: The heart was moderately enlarged to the left exhibiting the prominence and rounding of the left ventricular contour characteristic of hypertensive heart disease; the broad diameter of the heart was also increased. The thoracic ao ta was very tortuous and the arch was conspicuously widened. The ascending aorta and the knob were unusually prominent, as was the descending aorta which curved convexly to the left to reach the midline at the diaphragm. The lung fields were not remarkable.

Contrast roentgenogram (Fig. 14); frontal position at nine seconds: The pulmonary veins, the left ventricular cavity, the aorta and its branches at the arch were well filled. The left ventricular wall was thickened and measured

FIG. 13. A. W., aged fifty-five; blood pressure 245/145. Hypertensive heart disease. Conventional roentgenogram—frontal position. Note left ventricular enlargement and tortuosity of aorta.

1.3 cm. The ascending aorta at its origin was lost in the density of the spine but was well visualized in its mid-portion where it measured 3.5 cm. transversely. The innominate artery arising from the arch was enlarged and tortuous and exhibited "buckling" shortly above its origin. The left common carotid and subclavian arteries also were enlarged and tortuous. The aortic knob projected to the left and its wall was 3 mm. in width. The descending aorta below the knob measured 3.3 cm. in diameter. The ascending and descending portions of the aorta enclosed a clear oval space indicating the marked degree of unfolding of the thoracic aorta. The right supracardiac border was jointly formed by the unopacified superior vena cava and the ascending aorta; at no place did the aorta project farther than the right lower cardiac border.

Conventional roentgenogram (Fig. 15); left anterior oblique position (55°): The left ventricular contour of the heart appeared enlarged. The ascending aorta was dimly outlined and overshadowed by the breast. The transverse and descending portions of the aorta were dense permitting identification of the outer border, but the inner wall was poorly defined. There was obvious unfolding of the entire thoracic aorta.

Contrast roentgenogram (Fig. 16); left anterior oblique position (60°): The superior pulmonary veins, the left auricle, and entire aorta including its branches at the arch were outlined. The pulmonary veins seen in cross section lay above the left atrium which was well outlined and occupied the upper left quadrant of the heart. The left ventricle was not visualized probably because it was in contraction. The entire thoracic aorta was diffusely dilated and due to extreme unfolding it described almost a perfect "O" in its course. All the arterial branches from the arch were enlarged, particularly the innominate artery which shortly above its origin turned abruptly backward and then upward before dividing into its two branches. The tortuosity and "buckling" of this vessel is seen to best advantage in this view. The left subclavian artery as it passed upwards and posteriorly, formed the anterior side of the "aortic triangle"[40] which was bounded posteriorly by the spine and inferiorly by the transverse aorta.

Comment. Visualization gave the following detailed information in this case of hypertensive heart disease: The left ventricular cavity was moderately enlarged

FIG. 14. A. W. Hypertensive heart disease. Contrast roentgenogram—frontal position at nine seconds. LV = left ventricle; AO = aorta; IA = innominate artery. Pulmonary veins, left ventricle, and aorta filled. Note thickness of ventricular wall; outer border indicated by white arrow, inner by black line. Note tortuosity of aorta and "buckling" of innominate artery. Ascending aorta enclosed by parallel lines, arrow lies against outer wall of aorta.

and the wall was moderately thickened (Fig. 14); the thoracic aorta showed only slight enlargement in diameter (3.5 cm.), but the aortic loop was conspicuously rounded and tortuous probably as a result of the hypertension. The tortuosity is shown best in the frontal view in which the ascending aorta curves sharply to the right but curiously does not extend beyond the right cardiac border below. The transverse aorta forms the prominent aortic knob and then curves backward and downward to become the descending aorta which in turn describes a convex curve to the left as it passes to the diaphragm (Fig. 14). The left oblique projection presents the classical picture of unfolding of the aorta in which this vessel as a result of high internal pressure assumes a round instead of an inverted "U" shape in its course through the thorax (Fig. 16). The tortuosity or "buckling" of the innominate artery which was not evident on the conventional roentgenogram is clearly shown in both frontal and left oblique projections

Fig. 15. A. W. Hypertensive heart disease. Conventional roentgenogram—left anterior oblique position at 55°. Note prominence of left ventricular border and density and unfolding of aorta. White arrow indicates breast shadow which obscures ascending aorta.

Fig. 16. A. W. Hypertensive heart disease. Contrast roentgenogram—left anterior oblique position (60°) at nine seconds. Pulmonary veins (PV), left atrium (LA), and aorta (AO) filled. Parallel lines bracket ascending and descending portions of aorta. Arrow points to apex of "aortic triangle." Note O-shaped contour of thoracic aorta and dilated innominate artery (IA).

in which this vessel was seen to bend sharply to the right and then posteriorly before dividing into the right common carotid and subclavian arteries (Figs. 14 and 16). The left subclavian artery also is of particular interest since it obviously forms the anterior side of the "aortic triangle" (Fig. 16), a conclusion previously reached by Parkinson and Bedford[40] from roentgenologic and pathologic study but now demonstrated for the first time during life. The left atrium appeared entirely normal as did the pulmonary blood vessels. The remainder of the heart which is not described in this report exhibited no abnormality.

In summary, the following data regarding the left chambers of the heart and the aorta in this case of hypertensive heart disease were obtained: The left ventricle was moderately dilated and hypertrophied, while the left atrium and the right chambers were normal in size and shape. The

thoracic aorta itself was only slightly dilated but the aortic loop was conspicuously tortuous and rounded in its course through the thorax. The innominate, the left common carotid, and the left subclavian arteries were dilated and tortuous, the tortuosity being sufficiently marked in the first one to cause "buckling." The left subclavian artery was shown to form the anterior boundary of the "aortic triangle."

Fig. 17. E. L., aged eighty-three. Arteriosclerotic heart disease. Conventional roentgenogram—frontal position. Upper arrow lies immediately above calcified tubercle in the lung and points to calcified plaque in aortic wall. Middle arrow points to density adjacent to bronchus. Lowest arrow indicates apparent right border of the heart.

D. ARTERIOSCLEROTIC CARDIOVASCULAR DISEASE

Atherosclerosis of the coronary arteries may cause either generalized or local slight fatty degeneration of the heart muscle resulting in diffuse myocardial fibrosis, or it may lead to coronary thrombosis, myocardial infarction, and fibrosis of the involved area. In the former case, there is usually little or no change in size of the heart whereas cardiac enlargement is a common sequel of myocardial infarction. In the absence of coronary thrombosis and heart failure there may be advanced coronary artery disease without any detectable change in the size of the heart. Arteriosclerosis of the aorta is an independent process and unrelated to the myocardial disease which frequently accompanies it. Here the process begins as a deposition of fat in the intima followed by fibrosis and calcification of these areas and stiffening of the aortic wall. As a result of this sclerosis, elongation and tortuosity of the aorta develop.

The roentgenographic evidence of arteriosclerotic cardiovascular disease obviously depends upon the nature and degree of involvement of the heart and the aorta. The cardiac shadow usually remains normal in size and contour in the absence of myocardial infarction and circulatory failure, but when these occur they are followed by enlargement of the heart. Evidence of arteriosclerosis of the aorta consists of tortuosity of the vessel, widening of the aortic arch, elevation and prominence of the aortic knob, and calcification of the wall. These features may be observed in the frontal view but are usually much clearer in the left oblique projection.

In this patient, aged eighty-three, there was only a moderate degree of coronary artery and myocardial disease as indicated by the decrease in cardiac reserve and the electrocardiographic changes, but there was a more marked involvement of the aorta by the arteriosclerotic process. Hypertension was excluded as a factor in the cardiovascular disease by the absence of a positive history and clinical evidence of previous hypertension, the lack of left ventricular enlargement, and the normal blood pressure now. The conventional frontal roentgenogram (Fig. 17) showed an apparent widening of the cardiac shadow to the right and the left and also a widening of the supracardiac shadow. The latter was due to accentuation of the curve of the ascending aorta, which, however, did not surpass the right cardiac border, and to moderate convexity of the descending aorta toward the left. There was calcification of the aortic wall at the knob. In the left

oblique view (Fig. 20) the heart was not remarkable, but the aorta exhibited a moderate degree of dilatation and unfolding and a more marked degree of elongation and elevation of the arch. From these observations it was apparent that the patient had arteriosclerotic cardiovascular disease with enlargement of the heart and the aorta.

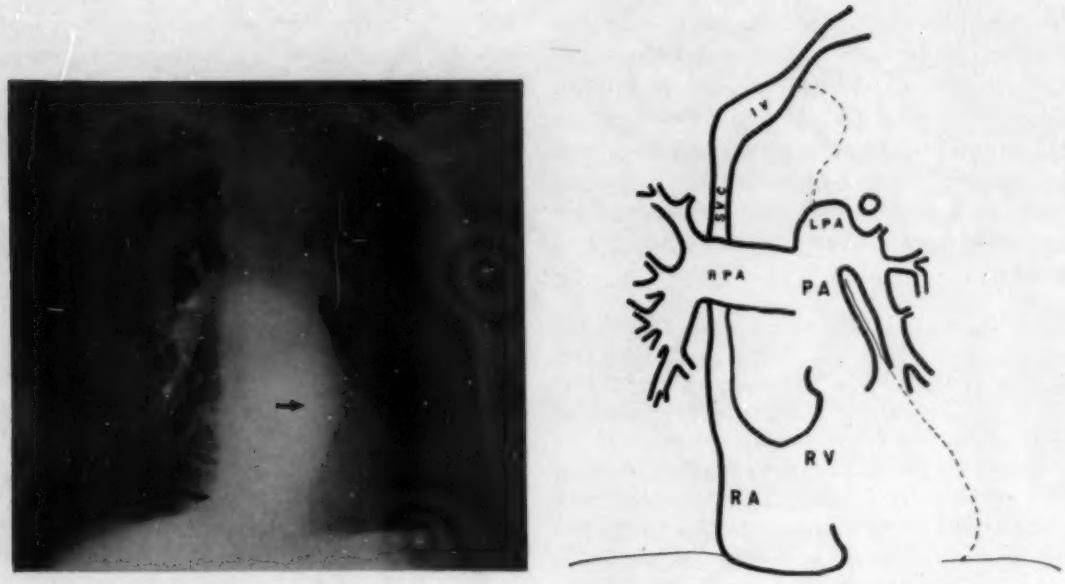
E. L., aged eighty-three. Conventional roentgenogram (Fig. 17); frontal position: The heart appeared to be enlarged to right and left, measuring 14.5 cm. transversely, and the supracardiac shadow was widened to 9 cm. at its widest diameter. The curve of the ascending aorta seemed to lie more medially than the right cardiac border. The aorta was elongated upward and to the left and exhibited a prominent knob which contained a calcium plaque. The descending aorta described a convex arc to the left and disappeared behind the cardiac shadow. The lung fields were clear except for obliteration of the left costophrenic sinus, thickening of the left axillary pleura and calcified tubercles in the upper third of each lung field. At the left hilum there was a small rounded opacity 9 mm. in diameter which lay immediately above and in close relationship to a small bronchus.

Contrast roentgenogram at five seconds (Fig. 18): The left innominate, the superior vena cava, the right atrium and ventricle, the pulmonary artery with its two main branches and their subdivisions were opacified. The left innominate vein crossed above the aorta to the right side of the spine where it entered the superior vena cava; this vessel in turn could be followed to the right atrium. The actual right border of the heart defined by the right atrium lay 2 cm. to the left of the right border of the spine which apparently formed the right cardiac border in the conventional film. The actual transverse diameter of the heart, thus, was 12.5 cm. instead of 14.5 cm. Only the diaphragmatic portion and the conus arteriosus of the right ventricle were visible, the middle portion being obscured by the density of the descending aorta. The left cusp of the pulmonary valve lying immediately above the conus indicated the point of origin of the pulmonary artery. This vessel passed obliquely backwards and upwards to divide into right and left branches at the left border of the spine, and these arteries in turn subdivided at each hilum. The wall of the pulmonary artery measured 2 mm. in thickness. The round shadow near the left hilum was distinctly more opaque than in the conventional film, confirming its arterial nature.

Contrast roentgenogram (Fig. 19); frontal position at sixteen seconds: The pulmonary veins, the left atrium, the left auricula, the left ventricle, and the entire thoracic aorta including the branches at the arch were visualized. The left auricula appeared as a tongue-like shadow which reached to the left border of the heart lying between the pulmonary arc and the left ventricular curve. The rest of the atrium could not be seen. In this roentgenogram the left ventricular wall was apparently greatly thickened, probably due to incomplete filling of the cavity, for in a subsequent film the wall actually measured only 1.1 cm. The beginning of the ascending aorta was ill defined, but the rest of the aorta was well outlined, the ascending, transverse, and descending portions measuring 4.2, 3.6, and 3.3 cm. respectively. The aortic curve on the right side reached to but did not exceed a vertical line drawn through the right cardiac border. The aortic wall was thickened (3.5 mm.) and the calcified plaque seemed to lie within it instead of on its inner surface. The right innominate and left common carotid and subclavian arteries were clearly outlined and appeared to be normal.

Conventional roentgenogram (Fig. 20); left anterior oblique position at 60°: The heart was not enlarged, but the thoracic aorta which contained calcified plaques in the arch was moderately dilated and unfolded; only the outer contour was clearly outlined.

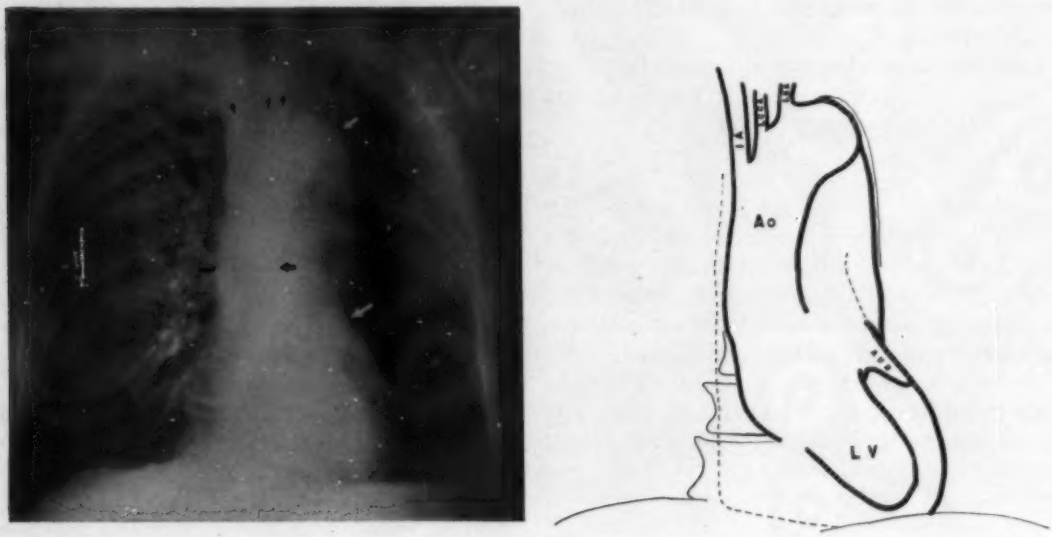
Contrast roentgenogram (Fig. 21); left anterior oblique position (60°) at seventeen seconds: The left atrium, the left ventricle, the aortic valve, and the entire thoracic aorta were identified. The left atrium occupied the superior and upper pole of the left side of the heart. The left ventricle lay below and anterior to it; the left ventricular wall was visible and measured 1.1 cm. in thickness. The aortic valve identified by two cusps and the corresponding sinuses lay to the right of the left atrium and at the upper pole of the left ventricle. As in the frontal view the ascending, transverse, and descending portions of the aorta measured 4.2, 3.6 and 3.3 cm. respectively, and the wall of the ascending aorta was 3 mm. in thickness. The innominate,



A *B*

FIG. 18. *A.* E. L. Arteriosclerotic heart disease. Contrast roentgenogram—frontal position at five seconds. Left innominate vein, superior vena cava, right atrium, right ventricle, and pulmonary arterial tree visualized. Lower arrow designates right atrium, upper arrow is against medial wall of conus arteriosus. Note relationship of right atrium and right border of spine; also opaque branch of pulmonary artery near left hilum seen in cross section.

B. Tracing of *A.* IV = innominate vein; SVC = superior vena cava; RA = right atrium; RV = right ventricle; PA = pulmonary artery; RPA = right pulmonary artery; LPA = left pulmonary artery.



A *B*

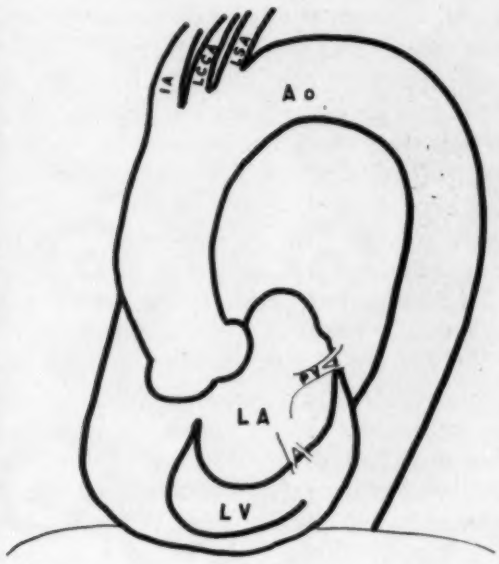
FIG. 19. *A.* E. L. Arteriosclerotic heart disease. Contrast roentgenogram—frontal position at sixteen seconds. Pulmonary veins, left atrium and auricula, left ventricle and thoracic aorta identified. Lower black arrows bracket ascending aorta and small arrows lie on aortic branches. Upper white arrow against outer wall of aorta. Auricula indicated by lower white arrow.

B. Tracing of *A.* AUR = left auricula; LV = left ventricle; AO = aorta; IA = innominate artery; LCCA = left common carotid; LSA = left subclavian artery.

the left common carotid and the left subclavian arteries were seen to arise from the anterior part of the transverse aorta and then to curve gently upwards and posteriorly. The left subclavian artery formed the anterior boundary of the "aortic triangle," the open space lying above the aortic arch and in front of the spine:

Comment. As in the other types of heart disease, visualization of the interior of the heart and the great vessels in this case of arteriosclerotic heart disease gave exact information which permitted accurate localization of the disease and evaluation of its degree. The entire right side of the heart together with the superior vena cava and the pulmonary arterial circulation, and the left atrium and auricula, were found to be normal (Figs. 18, 19 and 21). The left ventricle, although apparently hypertrophied in the frontal projection due to incomplete filling of the cavity (Fig. 19), actually was normal in size as shown in Figure 21 and in another frontal contrast roent-

Fig. 20. E. L. Arteriosclerotic heart disease. Conventional roentgenogram—left anterior oblique position at 60°. Aortic loop indicated by arrows. Note unfolding and lack of definition of aorta.



A *B*

Fig. 21. *A.* E. L. Arteriosclerotic heart disease. Contrast roentgenogram—left anterior oblique position (60°) at seventeen seconds. Left atrium, left ventricle, and aorta filled. Lower arrow indicates ventricular septum, small arrows the innominate, left common carotid, and subclavian arteries. Note distinctness of thoracic aorta, descending portion enclosed by arrows.

B. Tracing of *A.* PV = pulmonary vein; LA = left atrium; LV = left ventricle; IA = innominate artery; LCCA = left common carotid artery; LSA = left subclavian artery.

genogram not included in this study. The size and course of the thoracic aorta and the thickness of its wall are clearly shown in Figures 19 and 21. Contrary to the usual finding,[49] there is elongation of the aorta and moderate dilatation most marked in the ascending portion. The widening of the supracardiac shadow is due to uncoiling of the aortic loop and not to dilatation of the vessel itself. Further evidence of atherosclerosis of the aorta is provided by the calcification and the thickening of the wall.

In this patient visualization rendered a unique service by excluding the cardiac enlargement evident in the conventional roentgenogram. It established that the apparent prominence of the right cardiac border was caused by the deviated spine and not by the heart which was normal in size. The importance of overpenetration without opacification in roentgen examination of the heart is strikingly demonstrated in Figure 19 in which the right border of the heart and the spine are clearly defined although the right chambers no longer contain opaque material; whereas with the usual technique neither spinal nor cardiac border can be seen distinctly. Although it has long been known that blood vessels near the hilum may simulate calcified tubercles, proof in the individual case has been difficult if not impossible. However, the increase in the opacity of the structure in question during visualization of the pulmonary circuit (Fig. 18) proves its vascular nature beyond doubt.

In summary, visualization in this case study made the following contributions: (1) apparent enlargement of the heart was disproved by demonstration of the true cardiac borders; (2) the vascular nature of a rounded density near the left hilum which might have been called a calcified tubercle was shown; (3) the left atrium and ventricle were normal in size, shape, and location; (4) the thoracic aorta exhibited a moderate degree of dilatation, elongation, and unfolding with a moderate degree of thickening and calcification of the wall.

SUMMARY AND CONCLUSIONS

The results obtained by visualization of the chambers of the heart and the thoracic blood vessels in patients having rheumatic, syphilitic, hypertensive, and arteriosclerotic cardiovascular disease are reported and their value is discussed. Visualization in heart disease as in the normal gives a detailed picture of the cardiovascular system in which each major structure can be examined separately and its characteristics observed. As a result it has been possible to determine accurately the location and the degree of the gross pathological changes, thereby providing a means of solving certain hitherto perplexing problems.

The prominence of the pulmonary arc in the frontal view in our patients with rheumatic mitral stenosis and insufficiency was caused directly by the dilated pulmonary artery and not by the enlarged pulmonary conus or the left atrium; the left auricula formed the convexity immediately below the pulmonary arch. Elevation of the left bronchus was not caused by the left atrium but apparently by engorged pulmonary veins. In syphilitic aortitis, the intracardiac portion of the aorta, the most frequent site of involvement and one previously invisible, was outlined and the exact size, shape and location of aneurysms of the aorta were determined. The degree of left ventricular hypertrophy and dilatation, the tortuosity and unfolding of the thoracic aorta, the "buckling" of the innominate artery and the identity of the vessel forming the anterior boundary of the "aortic triangle" were demonstrated in hypertensive heart disease. In the patient with arteriosclerotic cardiovascular disease, the heart was found to be normal in size, the apparent enlargement in the conventional roentgenogram being due to the deviated spine, whereas the aorta exhibited a moderate degree of dilatation, elongation, and unfolding with calcification and thickening of the wall.

From these preliminary observations it seems apparent that inference regarding the condition of the heart which is based

upon grossly indirect evidence such as the alteration of the cardiac silhouette and the disturbance of adjacent structures can now be largely replaced by exact information obtained by separate visualization of the four chambers of the heart and the great blood vessels. As a consequence, the interpretation of conventional roentgenoscopy and roentgenography will become more accurate, enhancing the value of these widely used clinical methods of examination and ultimately making visualization of the chambers of the heart and the thoracic blood vessels necessary only in cases presenting unusually difficult diagnostic problems.

REFERENCES

1. Ameuille, P., Ronneaux, G., Hinault, V., DeGrez, and Lemoine, J. M. Remarques sur quelques cas d'artériographie pulmonaire chez l'homme vivant. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1936, *60*, 729–739.
2. Assmann, H. Die klinische Röntgendiagnostik der inneren Erkrankungen. Fifth edition. F. C. W. Vogel, Berlin, 1934.
3. Bardeen, C. R. Determination of the size of the heart by x-ray. Am. J. Roentgenol., 1917, *4*, 604.
4. Bedford, D. E., and Treadgold, H. A. Size of the healthy heart and its measurement. *Lancet*, 1931, *2*, 836–842.
5. Castellanos, A., Pereiras, R., and Garcia, A. Angiocardiography in the child. VII Cong. Assoc. Med. Pan American, Havana, 1938.
6. Castellanos, A., Pereiras, R., and Vasquez Paussa, A. On special automatic device for angio-cardiography. *Bol. Soc. cubana de pediat.*, 1938, *10*, 209–215.
7. Conte, E., and Costa, A. Angiopneumography. *Radiology*, 1933, *21*, 461–465.
8. Dann, D. S. The uncoiled aorta. Part I. The normal aorta. Am. J. Roentgenol. & Rad. Therapy, 1930, *23*, 358–372.
9. Dann, D. S. The uncoiled aorta. Part II. The pathologic aorta. Am. J. Roentgenol. & Rad. Therapy, 1930, *24*, 154–162.
10. Dietlen, H. Orthodiagraphische Untersuchungen über pathologische Herzformen und das Verkalten des Herzen bei Emphysem und Asthma. *München. med. Wchnschr.*, 1908, *55*, 1770.
11. Dietlen, H. Das Herz bei Emphysem und Bronchialasthma. Herz und Gefässe in Röntgenbild. Leipzig, 1923.
12. Egas Moniz, Lopo de Carvalho, and Almeida Lima. Angiopneumographie, *Presse méd.*, 1931, *39*, 996–999.
13. Evans, W. Course of the Oesophagus in Health, and in Disease of the Heart and Great Vessels. Medical Research Council. Special Report Series, No. 208. His Majesty's Stat. Off., London, 1936.
14. Eyster, J. A. E. Size of the heart in the normal and in organic heart disease. *Radiology*, 1927, *8*, 300–306.
15. Eyster, J. A. E. Determination of cardiac hypertrophy by roentgen-ray methods. *Arch. Int. Med.*, 1928, *41*, 667–682.
16. Forssmann, W. Ueber Kontrastdarstellung der Höhlen des lebenden rechten Herzens und der Lungenschlagader. *München. med. Wchnschr.*, 1931, *78*, 489–492.
17. Gray, H. Anatomy of the Human Body. Twenty-third edition. Lea and Febiger, Philadelphia, 1936.
18. Groedel, F. M. Die Röntgendiagnostik der Herz- und Gefässerkrankungen. Hermann Muesser, Berlin, 1912.
19. Hampton, A. O., Bland, E. F., and Sprague, H. B. Further studies of the aorta with special reference to luetic aortitis. *Am. Heart J.*, 1930, *6*, 77–81.
20. Hampton, A. O., and Jones, T. D. Clinical and roentgenological study of the aorta, with special reference to luetic aortitis. Am. J. Roentgenol. & Radium Therapy, 1930, *23*, 390–395.
21. Heim de Balsac, R., and Routier, D. Roentgenologic anatomic study of left auricle following post-mortem opacification. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, 838–847.
22. Heuser, C. Arteriografía directa, aortografía por impregnacíon del retículo endotelium, vasos grafías del pulmón por medio del sondeo de la aurícula derecha. *Rev. Asoc. méd. argent.*, 1932, *46*, 1119–1128.
23. Hirsch, I. S. Recording of cardiac movements and sounds by the roentgen ray (kymophonoroentgenography). *Radiology*, 1934, *22*, 403; 1934, *23*, 720.
24. Hodges, F. J., and Eyster, J. A. E. Estimation of transverse cardiac diameter in man. *Arch. Int. Med.*, 1926, *37*, 707–714.
25. Hodges, P. C., and Eyster, J. A. E. Estimation of cardiac area in man. Am. J. Roentgenol. & Rad. Therapy, 1924, *12*, 252–265.
26. Holzknecht, G. Die röntgenologische Diagnostik der Erkrankungen der Brusteingeweide. Hamburg, 1901.
27. Kirch, E. Das Verhalten von Herz und Kreislauf bei rechtsseitiger (pulmonaler) Herzhypertrophie. *Würz. Abhandl. a. d. Gesamtgeb. d. prakt. Med.*, 1925, *22*, 73.

28. Kirch, E. Pathogenese und Folgen der Dilatation und der Hypertrophie des Herzens. *Klin. Wchnschr.*, 1930, *9*, 769–772; 817–819.
29. Kirch, E. Der Entwicklungsablauf der rechtsseitigen tonogenen Herzdilatation bei Mensch und Versuchstier und seine physiologische Erklärung. *Virchow's Arch. f. path. Anat.*, 1933, *291*, 682–694.
30. Kurtz, C. M. Orthodiascopy. Macmillan Co. New York, 1937.
31. Laubry, C., Cottenot, P., Routier, D., and Heim de Balsac, R. Etude anatomo-radiologique du coeur et des gros vaisseaux par opacification. *J. de radiol. et d'électrol.*, 1935, *19*, 193; 561; 700; 1936, *20*, 65.
32. MacCallum, W. G. Textbook of Pathology. Sixth edition. W. B. Saunders Co., Philadelphia, 1937.
33. Moore, S. Body section radiography with the laminagraph in pulmonary disease. *Am. Rev. Tuberc.*, 1938, *38*, 538–556.
34. Moritz, F. Ueber orthodiagraphische Untersuchungen am Herzen. *München. med. Wchnschr.*, 1902, *49*, 1.
35. Nemet, G. Some clinical aspects of the radiology of the heart. *Med. Clin. North America*, 1932, *15*, 1383–1407.
36. Nemet, G., and Schwedel, J. B. Roentgenographic studies of right ventricle. *Am. Heart J.*, 1932, *7*, 560–573.
37. Nuvoli, I. Arteriografia dell'aorta toracica mediante puntura dell'aorta ascendente o del ventricolo s. *Policlinico (sez. prat.)*, 1936, *43*, 227–237.
38. Palmer, J. H. Development of Cardiac Enlargement in Disease of the Heart: A Radiological Study. Medical Research Council. Special Report Series, No. 222. His Majesty's Stat. Off., London, 1937.
39. Parkinson, J. Enlargement of the heart. *Lancet*, 1936, *1*, 1337; 1391.
40. Parkinson, J., and Bedford, D. E. Aortic triangle; radiological landmark in left (or II) oblique position. *Lancet*, 1936, *2*, 909–911.
41. Parkinson, J., and Hoyle, C. The heart in emphysema. *Quart. J. Med.*, 1937, *6*, 59–86.
42. Paterson, R. Value of roentgenologic study of esophagus and bronchi in cases of heart disease, especially mitral disease. Am. J. Roentgenol. & Rad. Therapy, 1930, *23*, 396–408.
43. Ravina, A. L'exploration radiologique des vaisseaux pulmonaires par l'injection de substances de contraste. *Progrès méd.*, November 3, 1934, pp. 1701–1705.
44. Reynolds, R. J. Cineradiography. Am. J. Roentgenol. & Rad. Therapy, 1935, *33*, 522–528.
45. Rigler, L. G. Visualized esophagus in diagnosis of diseases of heart and aorta. Am. J. Roentgenol. & Rad. Therapy, 1929, *21*, 563–571.
46. Robb, G. P. and Steinberg, I. Practical method of visualization of chambers of the heart, the pulmonary circulation, and the great blood vessels in man. *J. Clin. Investigation*, 1938, *17*, 507.
47. Robb, G. P., and Steinberg, I. Visualization of the chambers of the heart, the pulmonary circulation, and the great blood vessels in man. Am. J. Roentgenol. & Rad. Therapy, Jan., 1939, *41*, 1–17.
48. Robb, G. P., and Steinberg, I. Visualization of chambers of the heart, the pulmonary artery, and the great blood vessels in the normal; preliminary study. To be published.
49. Roesler, H. Clinical Roentgenology of the Cardiovascular System. Charles C Thomas, Springfield, Ill., 1937.
50. Routier, D., and Heim de Balsac, R. Modifications trachéales et bronchiques chez les mitraux. *Arch. d. mal. du coeur*, 1937, *30*, 861–870.
51. Rubin, E. L. Visualization of pulmonary artery by x-rays. *Brit. J. Radiol.*, 1937, *10*, 501–514.
52. Schwedel, J. B., and Epstein, B. S. Radiological study of pulmonary artery with special reference to main branches. *Am. Heart J.*, 1936, *11*, 292–302.
53. Smith, H. L. Relation of weight of the heart to weight of the body and of the weight of the heart to age. *Am. Heart J.*, 1928, *4*, 79–93.
54. Staehelin, R. Pathologie, Pathogenese und Therapie des Lungenemphysems. *Ergebn. d. inn. Med. u. Kinderh.*, 1915, *14*, 516.
55. Steel, D. Roentgenological and pathological findings in some of the valvular lesions. Am. J. Roentgenol. & Rad. Therapy, 1930, *23*, 384–389.
56. Steele, J. M., Jr. Compression and displacement of bronchi in mitral stenosis. *Am. Heart J.*, 1928, *4*, 53–58.
57. Steele, J. M., Jr., and Paterson, R. Distortion of bronchi by left auricular enlargement. *Am. Heart J.*, 1929, *4*, 692–708.
58. Steinberg, I., and Robb, G. P. Mediastinal and hilar angiography in pulmonary disease. *Am. Rev. Tuberc.*, 1938, *38*, 557–569.
59. Stewart, W. H., Hoffman, W. J., and Ghiselin, F. H. Cinefluorography. Am. J. Roentgenol. & Rad. Therapy, 1937, *38*, 465–469.
60. Stumpf, P. Das röntgenographische Bewegungsbild und seine Anwendung (Flächenkymographie und Kymoskopie). Georg Thieme, Leipzig, 1931.
61. Vaquez, H., and Bordet, E. Radiologie du coeur et des vaisseaux de la base. Fourth edition. J. B. Baillière et Fils, Paris, 1928.
62. White, P. D. Heart Disease. Second edition. Macmillan Co., New York, 1937.

DISCUSSION

Dr. George W. Holmes, Boston. I know that many men in the audience realize, as I do, that this is an epoch-making occasion. History has been made in cardiac diagnosis here today. I have never listened to a paper on the heart or read an article that to my mind offers more interest and more possibility for the future than the one to which we have just listened.

Dr. Carleton B. Peirce, Montreal, Canada. I think you all can appreciate what a real thrill I had when Dr. Steinberg told me in Los Angeles this June that he had something he would like to show me. With his characteristic modesty, he was not quite sure whether or not it would be interesting. I spent as much time as I had available then, and I wanted to spend hours and hours more. Dr. Hodges has spoken of the difficulties in measuring heart size. We all know the difficulties that we have had in analyzing the cardiovascular shadows. I am sure that Dr. Holmes is going to speak of very definite changes which this will permit in our interpretation of the change in the heart and major vessels in relation to the bronchi and lungs. The rather startling demonstration which Dr. Steinberg has given you of aneurysm of the pulmonary artery, and the emphasis on the fact that the junction between the pulmonary veins and the left atrium is a very broad and large space which, of course, becomes involved quite early in any obstruction to ready passage from the atrium into the ventricle on the left side as in mitral disease.

I wish to thank Dr. Robb and Dr. Steinberg for this opportunity to express my appreciation of this work.

Dr. Samuel Levine, Boston. I am sure that I would be expressing what is going on in the minds of clinicians in general in extending to Dr. Robb and Dr. Steinberg our great appreciation and congratulation on this epoch-making piece of work. This is really the first big advance in roentgen diagnosis of the heart since the x-ray was discovered, except for the refinements in technique of roentgen manipulation in films and chemical development, in roentgen tubes and so on. From the clinician's point of view, it seems to me that this is a new era.

It would be idle to speculate on how far this new development will go. This is the threshold of something new. It is extraordinary, when something is discovered, to see how minutely it can be used in the hands of expert roentgenologists. That has been the history of other developments in technique and one can imagine a great many things about this new approach.

The idea of measuring as accurately, as it now seems apparent, the thickness of various chambers, the position of the valves, possibly even intracardiac defects such as a defect in the ventricle septum; watching the pulmonary vessels even the minute ones; the distribution of this foreign opaque substance into the inner branches of the pulmonary vessels; the aid it may give us, therefore, in the diagnosis of peripheral pulmonary arterial disease—there is no end to what one can speculate about it. I know that we will all have to keep our eyes open for new bits of information, both in myocardial disease and vascular disease.

On the question of obstruction, the slide which showed the kinking of a vein impressed me very much. One can actually localize its position. We were not told here at this meeting but we were outside in the hall that 300 of these injections have been made without any mishap. I would like to ask if there are any contraindications. If nothing happens to 300 cases, we need only a few more to be fairly content that it is a safe procedure. I would like to ask whether coronary artery disease is a contraindication for injecting this foreign substance.

Some of the iodine preparations are very irritating. I hope you have not experienced it, but perhaps you have heard from other people's experience that the dye used in cholecystography can precipitate a coronary closure. I have seen that. I have had it happen to one of my own patients under my eyes, and I saw another one, so that it is a real thing. Yet I am told that this is a much more innocuous iodine preparation which very likely is not as irritating. I therefore would like to know if there is any contraindication at all to the injection of this substance.

I feel certain that we have all heard a tremendously important demonstration of a new principle, and beyond that, I do not see that I or any of us can add more in the way of comment. We should either start doing it or watch for further developments.

INFLAMMATORY DISEASES OF THE PERICARDIUM*

By EUGENE FREEDMAN, M.D.
From the Department of Roentgenology of The University Hospitals
CLEVELAND, OHIO

AMONG the three constituents of the heart, the endocardium, myocardium, and pericardium, the latter is the one least frequently subject to disease. Most of the diseases occurring in the pericardium are either inflammatory or traumatic in etiology. Tumors of the pericardium, primary or secondary, are extremely rare and are hardly to be considered from the standpoint of differential diagnosis.

The early roentgenologic literature contains ample communications concerning the diagnosis of pericardial effusions. Holmes[8,9] in 1920 and in 1924 published his experiences and results of his animal experiments, adding a valuable diagnostic sign to the previously known ones. His publications represent the roentgenologic summary of pericardial effusions and knowledge of the difficult problem has increased but little since.

The developments in the past decade in the field of the surgical exploration of the heart gave a new impetus to the roentgen study of the pericardium. Since the publication by Rehn[13] in 1920 of the successful removal of parts of the thickened pericardium with excellent clinical results, the disease known as cardiac compression due to pericardial scar (adhesive pericarditis) passed from the realm of symptomatic medical treatment into that of active surgical therapy. This development brought about a more generalized knowledge of the syndrome and an intensive search for more roentgen signs by which the condition may be recognized.

The experiences in the past years demonstrate that the roentgen signs of cardiac compression are more definite than those of pericardial effusion. It is generally recognized now that the roentgenologic examination enables one to exclude many cases suspected clinically and to establish a positive diagnosis of cardiac compression in each instance by careful correlation of clinical and roentgenologic signs.

PERICARDIAL EFFUSION

Despite the numerous roentgen signs of pericardial effusion, its differentiation from other heart conditions and its recognition at an early stage are still difficult. While dry pericarditis does not give any roentgen signs, effusion produces definite changes. As to the amount of fluid necessary to produce changes in the cardiopericardial shadow, the opinions are varied. Amelung[1] considers 400 cc. as the minimum amount, while Roesler[14] thinks that in an adult 250 cc. will produce recognizable roentgen signs. He states that in infants as little as 10 cc. has been diagnosed.

Sauerbruch[15] found that fluid first collects in the region of least resistance, in the lower pericardial recesses. Fleischner,[6] examining patients with suspected early pericardial effusions, was able to confirm roentgenologically Sauerbruch's clinical observation. In subsequently proved cases he found that while the heart shadow did not seem to be enlarged, its posterior lower border possessed a convex bulge instead of the normally straight oblique or slightly concave contour. He considers this sign as the only important one in early effusions.

As the fluid increases in amount, marked changes in the configuration occur. The transverse diameter increases, the normal arches become obliterated and the heart shadow becomes globular or bag shaped. If the exudation is large, it pushes the upper anterior pericardial recesses upward and a shortening of the vascular shadow

* Read in a Symposium on Heart Disease at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N.J., Sept. 20–23, 1938.

occurs. The heart then resembles a round bottle with a short neck, due to the fact that the margin of the distended pericardium juts outward immediately below the shadow of the great vessels and assumes an arcuate shape. The bulging is more marked on the left side than on the right. Thus, the transverse diameter of the heart becomes greater than the longitudinal.

The pulsations are at first diminished; later they may become undulatory or entirely imperceptible.

There has been considerable discussion as to the visibility of the heart within the pericardial fluid. Schwaer[17] was the first who reported the so-called "kernel shadow," a dense, centrally located nucleus, supposedly due to the heart itself. Schultze[16] in 1921 reported a similar finding. As recently as 1935, Cola[5] reported a case in which the roentgenogram showed the difference between the central nucleus and the surrounding exudate. Pericardotomy yielded abundant fluid and no air was present in the pericardial cavity.

Assmann,[2] however, failed to observe this sign with certainty in a large series of patients. Holmes,[8] injecting ascitic fluid in dogs' pericardial cavities, failed to see a "kernel shadow" in any of his experiments. Kloiber and Hochschild[11] likewise deny the visibility of the heart within the pericardial effusion, publishing a case where a paravertebral abscess led to the production of a "double cardiac shadow."

The cardiodiaphragmatic angle, despite the clinical conception of its obtuseness, remains acute in the majority of cases because the pericardium is unable to distend as much at its point of fixation to the diaphragm as it does above. However, the acuteness of the cardiodiaphragmatic angle may be influenced by an elevated or immobile diaphragm or by effusion at the basal portions of the pleural cavity. Under such circumstances the cardiodiaphragmatic angle may be found to be obtuse.

From a differential diagnostic standpoint, large hearts due to myocarditis or to decompensated chronic valvular disease may cause difficulties. They may assume the form of a flabby bag or water bottle with obliteration of the normal arches and may exhibit diminished pulsations. An important diagnostic sign described by Holmes[9] is based on the changes of the cardiac silhouette during changes in the position of the patient. In the erect position the basal part of the pericardium yields to the pressure and the greatest width of the shadow moves toward the diaphragm. In the prone recumbent position with the head low and the abdomen elevated, the shadow of the great vessels becomes wider and the lower part of the heart narrower. The heart thus becomes more rectangular. The change in the configuration of the heart is very slight in cardiac dilatation. This sign is of great value, provided the patient is not too orthopneic, rendering the prone position impossible.

The injection of air into the pericardial cavity has been recommended after withdrawal of fluid to demonstrate the actual size of the heart. The heart is seen to be surrounded by air, the outer boundary of which is formed by the well visualized pericardium. The fluid level below the air shows the transmitted pulsatory and undulatory movements. Naturally, this is a rather complicated and difficult procedure to carry out and its practical importance is consequently slight.

The most reliable differential diagnostic method is the examination of the heart at frequent intervals, and the finding of considerable change in the size of the heart from normal to an enlarged one, and vice versa, within a short period of time. This is more likely to happen in pericardial effusions than in dilated hearts.

The kymographic examination, as was utilized until recently in pericardial effusions, has not been of great aid in the differentiation because it demonstrated only decreased or hardly discernible pulsations, types that occur in decompensated hearts, in myocardial damage, as well as in pericarditis. The kymographic sign recently described by Berner[4] appears to be of great

promise and deserves further investigation.

He bases his sign on the fact that the pulsatory amplitude of the left cardiac contour (aorta and left ventricle) is equal. He found a discrepancy of the pulsatory excursion of the left side in pericardial effusions. The pulsations were vigorous in the region of the aortic knob and were decreased in amplitude in the left ventricular region, due to the fact that nothing prevents the kymographic recording of the pulsations of the part of the aorta lying above the pericardial fold. He demonstrates the records of a patient where there was a discrepancy between the aortic and left ventricular pulsations during the presence of a pericardial effusion, while after the disappearance of the fluid the pulsations became equal in both regions. This sign is of no value in cases in which the aortic knob is calcified.

INFLAMMATORY DIVERTICULA OF THE HEART

The roentgen signs of the infrequently found encapsulated pericardial effusion were first described by Kienböck and Weiss[10] in 1929. Anatomically the disease is a combination of pericardial scar tissue formation with a localized encapsulation of a persisting exudate, usually on the right side. There is a sharply defined hexagonal, lobulated or semicircular bulging in the region of the encapsulation. The remaining part of the heart may be normal in size or enlarged. The structure is to be differentiated from mediastinal or lung tumors, aortic aneurysms and encapsulated mediastinal pleural effusion.

CARDIAC COMPRESSION DUE TO PERICARDIAL SCAR

This disease is known by several names, the most common being Pick's disease, or Pick's syndrome, originating from the name of the author who gave a complete description of it in 1896. Adhesive pericarditis and chronic mediastinopericarditis are other commonly used terms. Because in a great number of cases the pericardium fails to show evidence of acute or chronic inflammation but is changed into a hyalinized connective tissue scar, and because the symptomatology is not due to the scar itself but to its compressing effect, Beck[3] applied a new term to the condition. His proposed term, "cardiac compression due to pericardial scar" is both anatomo-pathologic and physiologic and consequently is more descriptive.

The condition represents either the sequelae or the end-results of a previous inflammation of the pericardium. The etiologic factor can be determined only in those few instances where it is due to a tuberculous process. Of the 26 cases operated upon by Beck, in only 4 was the condition found to be due to active tuberculosis. In the remaining ones histologic examination disclosed no clue as to the possible etiologic factor.

The pericardium is usually transformed into a firm, solid layer, the thickness of which measures anywhere between 2 and 26 mm. Due to adhesions between the parietal and visceral pericardium, the pericardial cavity is more or less completely obliterated. During the operation it is usually difficult to find the line of cleavage between the two layers and often blunt dissection is necessary in order to separate the pericardium from the heart. In addition, the pericardium is adherent to the neighboring organs. The obliteration of the pericardial cavity with thickening of the pericardium and the formation of external adhesions were present in each of our surgically treated cases.

The condition may occur in any decade of life with the exception of extreme youth and old age. It is most prevalent during the third, fourth, and fifth decades, in the order mentioned, 19 of the 26 cases having occurred in this age group. In our series it was more commonly found in males than in females, the ratio being 19 to 7, almost 3 to 1. Although colored patients represent 30 per cent of all admissions to our clinic, only 7 per cent of patients suffering from

cardiac compression occurred in this race (2 in 26).

The roentgen signs are based (1) on the effect of the scar on the configuration, size and pulsatory excursions of the heart, and (2) on changes caused by adhesions between the scar and the neighboring organs. The scar itself is not visualized except in those relatively rare instances when it becomes partially or completely calcified (armored heart).

One of the most common observations is the discrepancy between the clinical symptoms of decompensation and the absolutely or relatively small size of the heart. Among the 26 hearts, 18, or 65 per cent, were either normal in size or smaller than normal. In the remaining 8 cases, the cardiopericardial shadow was moderately enlarged in 6 cases and greatly enlarged in 2 cases. This finding is in accordance with the findings of McGuire and Hauenstein[12] who reviewed 67 cadavers in which various types of chronic inflammatory diseases of the pericardium were present. They found that adhesions binding the visceral and parietal pericardium together and to the neighboring organs are associated with cardiac hypertrophy only in instances when complicating hypertension or some valvular lesion coexists. Consequently, if the cardiopericardial shadow is enlarged, it should indicate either an underlying valvular or myocardial disease, or an extensive thickening of the pericardium.

Of our 26 patients examined, 6 came to autopsy and in each of the cases the weight of the heart was found to be within normal limits. Yet, in one of these patients, the roentgen examination showed an enlargement of the cardiopericardial shadow. However, the thickness of the pericardium measured between 6 and 26 mm. at various sites, explaining the enlargement.

As far as the configuration of the heart is concerned, the findings can be divided into three main groups. Group 1: the triangular with straight oblique left and right borders. Group 2: the globular with arcuate left and right borders. Group 3: the hearts with a mitral or an aortic configuration. There were 13 triangular, 8 globular and 5 of aortic or mitral configuration. Consequently, the triangular type can be considered the most typical, the globular being second in frequency.

The normal subdivision of the left and right arches is almost always absent in the triangular and globular hearts, while the remaining ones show them to persist, although not as definitely demarcated as in normal hearts. In many instances, dense adhesion bands are seen to extend from the lower border of the cardiac silhouette to the diaphragm.

Another sign that is seen in the majority of cases is the unchanged configuration of the heart shadow during the two phases of respiration, due to the rigidity of the thick and inelastic pericardium.

The pulsations are either absent or markedly decreased in amplitude. Occasionally the pulsations are normal, or of increased amplitude, over certain sections of the heart, mainly in the region of the left ventricle. These pulsations are in distinct contrast to diminished pulse waves in the region of the great vessels. Roesler[14] noted similar findings and thinks that "the increased amplitude does not express an increased stroke, but merely corresponds to an altered mode of ventricular contraction." According to him, "when the base of the heart is fixed, in the course of obliteration of the pericardial cavity, the longitudinal contraction of the ventricular cone is interfered with and this reduction in movement is compensated for by increased marginal excursions." Pulsatory excursions are occasionally found in the region of areas of calcification and the excursions of calcified plaques were well demonstrated on several kymograms.

The kymographic examination was found to be of no specific differential diagnostic value. The importance of this procedure lies in the fact that it represents a visual recording of the preoperative pulsations which can be compared any time with subsequent postoperative kymograms and

thereby the degree of improvement of the pulsatory excursions can be determined.

The presence of extrapericardial adhesions and the fixation of the heart are observed under the roentgenoscope by turning the patient to the left and to the right side, but more conclusively by taking postero-anterior roentgenograms during inspiration and expiration with the patient lying either on the left or right side. Normally, in this position, the heart moves during expiration toward the side on which the patient lies, while in inspiration it returns to the midline. If there is mediastinal fixation, this excursion is either diminished or absent.

The adhesions to the sternum are observed in the lateral position during inspiration and expiration, as described in a previous communication.

Another diagnostic sign described in a previous communication by the author is the obliteration or marked flattening of the aortic knob. This is the most consistent sign among all and was present in 21 of the 26 cases examined.

The superior vena cava was enlarged in over half of the instances. Of all the patients examined, the lungs and pleural cavities were found to be normal in five. Pleural effusions of various amounts, either unilateral or bilateral, was present in 16 patients and chronic passive hyperemia of the lungs in nine. Chronic pulmonary tuberculosis was found in 3 instances and silicosis in one. The diaphragm was normal in contour only in 6 patients. In the remaining ones the diaphragm was either obliterated by fluid or had irregular flattened contours due to pleurodiaphragmatic or pericardiodiaphragmatic adhesions. Interference with normal excursions of the diaphragm was frequently found.

Calcification of the pericardium was demonstrated in 9 of the 26 patients. In 2 additional instances, minimal amounts were found at operation but could not be detected on roentgen examination. The calcium deposits lie chiefly between the two leaves of the pericardium. Necrotized fibrin particles originating from the previous pericarditis become impregnated with calcium in the form of small needles, platelets or granules, which are loosely connected and freely movable against each other. This loose connection explains the fact that areas of calcification may show definite pulsatory excursions under the roentgenoscope and the kymogram. The amount of calcification varies from a few platelets to a complete encasing of the heart. Calcification is most frequently found in the region of the left atrium, and in the region of the descending ramus of the left coronary artery on the anterior surface of the heart.

Postoperative Changes. Marked change in the configuration of the heart after operation was never found. In several patients the heart shadow appeared to be somewhat larger after operation, due to the removal of parts of the pericardium, whereby the constricting effect of the surrounding ring was interrupted and the heart was allowed to dilate to its previous size. This effect was well demonstrated, mainly in the small triangular hearts, in which the formerly straight oblique borders changed into shallow convex arches. Due to the fact that the pericardium was removed mainly from the anterior surface of the heart, and only to a lesser degree from the lateral aspects, no actual decrease of the cardiopericardial shadow was noticed in any of the cases. The pulsations invariably showed an increased amplitude either throughout the entire circumference or of certain sections of the heart and these changes were quite well demonstrated by kymographic examinations.

CONCLUSIONS

1. Pericardial effusion leads to the formation of cardiac configurations which have been described by various authors as bag- or pear-shaped, or resembling a round bottle with a short neck. None of these configurations, however, are typical of pericardial effusion. They may be produced by decompensated hearts also.

2. The best differential diagnostic sign

between a decompensated heart and pericardial effusion is the one described by Holmes. This is based on the change of cardiac configuration during changes in the position of the patient. Unfortunately this procedure cannot be utilized in patients who are orthopneic.

3. The kymographic examination may be helpful in differentiating pericardial effusions from decompensated hearts. Berner observed vigorous pulsations of the aortic knob and decreased pulsations of the left ventricular region on the kymogram of a patient suffering from pericardial effusion. After the effusion disappeared the kymographic recording of the pulsatory amplitude of the left ventricle and aortic knob became equal.

4. The recognition of early pericardial effusion may be facilitated by finding an abnormally convex bulging of the posterior surface of the heart either roentgenoscopically or on the lateral roentgenogram (Fleischner).

5. The most reliable method is the daily roentgen examination of the heart and the finding of considerable change in its size within short periods of time.

6. Kienböck's inflammatory diverticulum of the heart represents an encapsulated pericardial effusion, most commonly on the right border. The remaining part of the pericardial cavity is usually obliterated.

7. Cardiac compression due to pericardial scar formation is usually represented on roentgen examinations by a normal-sized or smaller than normal heart. Moderate or marked enlargement of the cardiopericardial shadow is uncommon and may be due either to an unusually marked thickening of the pericardium or to an underlying valvular or myocardial disease.

8. The majority of the compressed hearts are either triangular or, less commonly, globular. In rare instances a compressed heart may show a mitral or aortic configuration.

9. The cardiac pulsations are usually diminished or absent. Occasionally, however, certain sections of the compressed heart may show vigorous pulsations.

10. The aortic knob is flattened or completely obliterated in the majority of compressed hearts.

11. Due to the marked thickening of the pericardium, the heart becomes rigid and in the majority of cases its configuration remains either unchanged or changes but little during the two phases of respiration.

12. The adhesions between the heart and pericardium lead to a fixation of the heart. The degree of fixation is determined with the aid of expiratory and inspiratory posteroanterior roentgenograms taken with the patient lying on either side and with lateral views taken during both inspiration and expiration.

13. The most conclusive sign of cardiac compression, the calcification of pericardium, is present only in the minority of instances (in 9 among 26 patients).

14. The kymographic examination is of no differential diagnostic value in cardiac compression. It only serves as a record which enables one to compare the difference between the pulsatory amplitude before and after the removal of the pericardial scar.

REFERENCES

1. Amelung, W. Zur Frage der doppelten Konturierung des Herzschattens im Röntgenbilde bei Perikarditis. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1921–1922, *28*, 519–524.
2. Assmann, H. Die Röntgendiagnostik der inneren Krankheiten. F. C. W. Vogel, Leipzig, 1922.
3. Beck, C. S. Two cardiac compression triads. *J. Am. M. Ass.*, 1935, *104*, 714–716.
4. Berner, F. Ein neues Röntgensymptom bei Herzbeutelerguss. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1937, *56*, 536–540.
5. Cola, G. Osservazioni radiologiche nelle pericarditi acute e croniche (cardiofibrechia). *Radiol. med.*, 1935, *22*, 125–146.
6. Fleischner, F. Perikarderguss. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1928, *38*, 402.
7. Freedman, E. Roentgenological diagnosis of cardiac compression due to pericardial scar. Am. J. Roengenol. & Rad. Therapy, 1937, *37*, 739–755.
8. Holmes, G. W. Radiographic findings in pericarditis with effusion. Am. J. Roentgenol., 1920, *7*, 7–15.

9. Holmes, G. W. Some observations on use of roentgen ray in diagnosis of pericarditis. *J. Am. M. Ass.*, 1924, *83*, 1745–1747.
10. Kienböck, R., and Weiss, K. Ueber das entzündliche Perikard-Divertikel. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1929, *40*, 389–418.
11. Kloiber, H., and Hochschild, H. Zur Frage des röntgenologischen Sichtbarwerdens des Herzens im Perikardialerguss. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1919–1921, *27*, 473–488.
12. McGuire, J., and Hauenstein, V. Diagnosis and treatment of concretio cordis: accretio cordis. *Ohio State M. J.*, 1937, *33*, 268–271.
13. Rehn, L. Ueber penetrirende Herzwunden und Herznaht. *Arch. f. klin. Chir.*, 1897, *55*, 315–329.
14. Roesler, H. Clinical Roentgenology of the Cardiovascular System. Charles C Thomas, Springfield, Ill. 1937.
15. Sauerbruch, F., and O'Shaughnessy, L. Thoracic Surgery. Revised edition. Edward Arnold & Co., London, 1937.
16. Schultze, F. Pericarditis exsudativa im Röntgenbilde. *Deutsche med. Wchnschr.*, 1921, *47*, 863.
17. Schwaer, G. Ueber Perikarditis exsudativa im Röntgenbilde. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1917–1918, *25*, 24–25.

DISCUSSION

Dr. George W. Holmes, Boston. Dr. Freedman has kindly referred to some experimental work which we did nearly twenty years ago. This work was undertaken primarily to determine whether or not it was possible to visualize the heart within the fluid-filled pericardium, and at that time there was considerable difference of opinion among roentgenologists on this point. I was assisted in that work by Dr. Charles L. Martin now of Dallas, Texas. One experiment which we carried out, not described by Dr. Freedman, seems to me to be so conclusive that I would like to state it here. A freshly removed dog's heart was suspended by threads in a pasteboard box coated with paraffin. The box was filled with solutions of varying specific gravities and roentgenograms made. In none of the experiments was the fixed heart visible unless the specific gravity of the fluid surrounding the heart was considerably higher or lower than is ever found in the body cavity. We reasoned that, if a fixed, immovable heart could not be seen, it was very unlikely even with improved technique that the living moving heart would ever become visible. I have yet to see a proved case. Cases reported, so far as I am able to determine, have either been due to fluid in the mediastinum or a mistaken interpretation.

The changes in the posterior aspect of the heart with change in shape of the posterior angle were, I think, first described by Williams. I should not be given the credit for that. In Boston we call it Williams' sign.

Dr. Freedman has called attention to the widening of the base of the heart shadow, with change in the position of the patient, which we emphasized at that time.

Since then, we have found one source of error in this sign. Some of the cases, particularly those with tuberculosis, have adhesions in that region and fluid in the lower part of the sac; in these cases, of course, no change in shape based on the change in position will be shown.

Dr. Freedman rightly devotes the major portion of his paper to an excellent discussion of the roentgen findings in the adherent pericardium. When we were working on that subject, we were not able to get very far in the diagnosis of this type of cardiac lesion. Since then, there have been great advances in diagnosis and it is becoming increasingly important because of the advances in surgical technique in cardiac disease. Roentgenologists are now asked to make that diagnosis much more often than they ever were before.

Dr. Samuel Levine, Boston. I might spend a moment to add a word concerning gallbladder dye injection and its relation to coronary artery catastrophies. I do not know much about it except from actual experience. I do not know the chemical phenomena involved nor of any particular predilection of that dye for the coronary arteries. I do know that in two instances directly, one during the injection and one directly after the injection of the dye, patients developed a classical acute coronary thrombosis, and one of them died a few weeks after the injection. There is no doubt that we were responsible for the sequence of events; it was not a coincidence.

Foreign irritating substances are dangerous to persons who have coronary arteriosclerosis. Whether an actual thrombosis results from the foreign substance going through the coronary arteries or whether a reaction takes place which produces coronary spasm, and that ruptures a subintimal plaque, according to the pathological studies of Dr. Timothy Leary, I do not know, but the clinical sequence is quite clear.

The moral that I have drawn from it is that I will give no intravenous dye in studying the gallbladder to any patient who, I recognize, has had angina pectoris or gross coronary artery disease. I would be content with the oral administration. I have seen no ill effects from that. I will not give intravenous gallbladder dye to any patient who has coronary pathology of which I am aware.

The diagnosis of pericardial effusion is difficult in the hands of the clinician in many instances, and it is not a simple thing even when we have the aid of the roentgenologist. I do not mean that we do not make adequate diagnosis but there are errors that we do make, errors in both directions, when everything points to a dilating heart and the pericardial cavity contains 500 cc. of fluid, and vice versa; even when the roentgen report points to fluid and it is all dilatation. I had that second experience which resulted in a fatality. I punctured a pericardial sac because the patient looked poorly. Probably the patient would have died within a week or two anyway, but he was going downhill and the configuration of the heart and the physical features pointed to a pericardial transfusion. If a large amount of fluid had been responsible for the serious situation, we could have relieved it by removing the fluid. I happened to hit a coronary vessel and he died of tamponade, with 400 cc. of blood in the pericardium. Autopsy showed nothing but this big blood clot which we had produced by nicking a vessel. The moral of that is that a pericardial tap is a dangerous procedure. Fortunately, it is very rarely necessary to tap a pericardial effusion. They do well if left alone. They may be very sick with a huge effusion and they can still do well and clear up entirely.

There are rare times when a pericardial tap is life saving, and it is a difficult decision for the clinician to make in deciding when the danger is greater—not to tap than to tap. The danger probably would be nil if fluid were present, but we still have the difficulty in differentiating a dilated, atonic heart that can have the same globular appearance and the same small pulsation as a pericardial effusion. The rapid change in size is helpful, both when determined clinically and when determined by the roentgenogram.

A word may be said about pericardial constriction. This has become an important subject, not because it is so common but it is all important for the few who have pericardial constriction. It is the one kind of heart disease, or one of the few types of heart disease, which the surgeon can help a great deal, and in many cases actually cure, so that it becomes incumbent upon the physician at least to be looking for this condition and to seek the aid of the roentgenologist to clinch the diagnosis.

I say clinch the diagnosis. The diagnosis really is a clinical one because we are more concerned with whether the pericardial disease is producing dynamic constriction of the circulation than whether adhesions are there or calcification. We occasionally see calcified pericardiums but no evidence at all of pericardial constriction. The pericardial constriction connotes that there is a difficulty in diastole of that heart, a difficulty of that heart in filling; that the blood cannot get into that heart very well; it cannot dilate. Some of them can contract very well, if they could only expand; if they would be permitted to fill they would be able to contract, because there is no inherent defect in the muscle or valves. The constriction is often around the right side of the heart so that it cannot fill or dilate.

There are a great many cardiacs, especially rheumatics, who come to autopsy who have pericardial adhesions. These adhesions are immaterial as far as the circulation is concerned. They are not the ones in which we are interested; they are not the ones the surgeon is going to help. But they are the ones that have large hearts. In other words, the type for which we are looking is the small heart which has no valve disease; on the whole, a hypotensive individual who has venous obstruction. The venous pressure is increased. They present themselves to the physician often with a picture resembling cirrhosis of the liver, but a simple distinction between the two is that in cirrhosis of the liver, there is portal obstruction and increased pressure below the diaphragm. In pericardiac constriction, there is increased pressure in the superior vena cava, in the arm as well as in the leg, and the increased venous pressure in the arm is of considerable help in distinguishing these cases.

We should keep in mind that from a therapeutic point of view, we want to help the dynamics of the circulation. You can therefore have pericardial adhesions in rheumatic hearts without any pericardial constriction. You can have pericardial calcification without any embarrassment of the venous return. You therefore not only need to incriminate the peri-

cardium but you also have to incriminate it in a mechanical sense; that is, it must be shown to obstruct the return flow or the systolic output. Primarily it is the venous return that is embarrassing and that is the main thing that is corrected by the surgeon.

These patients, I would like to repeat, have no inherent disease of the heart muscle. The coronary arteries are all right; the valves are all right. They are persons whose hearts sound innocent but have a great deal of evidence of congestion. They have a regular heart with no murmurs, no systolic murmur, no elevation in blood pressure, and yet present the picture of congestive failure. They are innocent-sounding hearts in which the pericardium is the cause of the mischief. I think the roentgenologist will be in a position to detect early clues. The physician may find the roentgenologist of great assistance. He may not be able to do venous pressure determinations but if he thinks about pericardial disease the roentgenologist may start him on the right track.

Vol. 42, No. 1

ROENTGENOLOGICAL ASPECTS OF ACQUIRED VALVULAR HEART DISEASE*

By MERRILL C. SOSMAN, M.D.

BOSTON, MASSACHUSETTS

ACQUIRED valvular heart disease in New England is largely due to rheumatic fever. Valvular heart disease is the cause of 40 per cent of the cardiac difficulties seen in our clinic. Hypertensive heart disease and coronary artery disease are next in frequency, with syphilis relatively infrequent. The valves most often involved are, in order of frequency, mitral, aortic and tricuspid. The pulmonary valve lesions are almost always congenital in origin and will be considered by Dr. Roesler with that group. Combined valvular disease is more common than single valvular lesions, as will be pointed out later.

The roentgenological characteristics of each valve lesion will be considered separately. In general, there are three important basic facts to remember and to establish before we consider the individual lesions: (1) The roentgenological method of examination is most valuable when the physician is trying to decide whether or not heart disease is present. In a case suspected of having heart disease the demonstration of cardiac enlargement or of abnormal cardiac contour practically always indicates heart disease. The converse is not true. Serious heart disease can and does exist in hearts which are normal in size and shape (and they may also be normal by all other methods of examination). The best example of this is coronary artery disease. The roentgenological method of examination is less useful and less important in advanced valvular heart disease, where the diagnosis is quite definite clinically. Its value here is only confirmatory or for matter of record.

(2) The second basic consideration to be emphasized is that the relative value of a given roentgenological examination is inversely proportional to the skill and judgment of the referring physician. Those roentgenologists who do not have physicians well trained in cardiology to work with can contribute more toward the establishment of the diagnosis of heart disease than those whose patients have been very accurately and carefully studied by a first-class cardiologist. The attitude of the roentgenologist, however, should be cooperative, remembering that the roentgenological is only one of a number of methods of physical examination.

(3) The third basic principle is more specific and is the well-known fact that valvular disease of the heart sooner or later results in dilatation or hypertrophy of one or more chambers.* From the enlargement of a certain chamber and the resultant change in the contour of the heart one can deduce which valves are at fault. There are other accessory findings which will be considered later, but the primary consideration should always be a determination of particular chamber enlargement.†

The frequency of rheumatic valvular heart disease varies greatly with the geographical location. It is quite frequent in the northern half of the United States and in similar temperate zones of Europe and England. It is rare in the southern half of the country and in tropical and subtropical countries. The relative frequency of the various valves involved is about the same wherever rheumatic fever exists. In 738 cases of rheumatic valvular heart dis-

* Using experimental valve lesions in animals, the change in contour occurs quite promptly. It may be delayed in human hearts as the progressive deformity of a valve may be very slow in developing.

† For a critical analysis of the value of the roentgenological method in study of cardiac enlargement, the Lumleian Lecture by Parkinson[3] is recommended.

* From the Department of Roentgenology, Peter Bent Brigham Hospital, Boston. Read in a Symposium on Heart Disease at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938.

ease in the Peter Bent Brigham Hospital, the *clinical* diagnosis (including autopsies) of valvular involvement was as follows:—

Combined mitral and aortic	361
Mitral alone	219
Aortic alone	158
Tricuspid alone	0
	738
(Tricuspid, organic and relative, with mitral or aortic or both)	189

This is about the same percentage frequency as reported by other authors, except for the large number of pure aortic valve involvement which will be discussed under that heading. An average of several groups reported by White,[13] Parrot,[4] Cabot,[1] Sperling[6] and others is:

Mitral alone	45%
Mitral and aortic	30%
Aortic alone	17%
All others	8%

The discrepancy in the figures concerning combined valvular disease would indicate that it is more difficult to diagnose accurately the involvement of two or more valves than of one valve only. Ordinarily one valve will be involved first, the other later and perhaps to less degree, so that we may be able to distinguish *predominant* and associated or secondary degrees of involvement. Calcification of one valve, the other being uncalcified, would suggest that the calcified valve was involved first.

MITRAL VALVE

Disease of the mitral valve practically always follows rheumatic fever, and results in dilatation of the left auricle. It is found most commonly between the ages of ten and thirty and involves the female sex more often than the male in a ratio of about 3 to 2. Mitral valve disease can be diagnosed by the general medical staff in 75 per cent of the cases and by the expert cardiologist in 93 per cent of the proved cases, according to the figures of Thompson and Levine.[10] Roentgenological assistance could therefore be of help and perhaps of critical value in one-fourth of the cases. The clinical signs, symptoms, and findings may be quite definite and the roentgenological findings inconclusive or negative; more often they agree, with the roentgenological findings confirmatory. Occasionally the clinical findings may be indefinite or negative, and the findings by roentgenoscopy and films quite definite.

The characteristic appearance of mitral valve disease is dilatation of the left auricle. This applies equally to mitral stenosis and mitral regurgitation, but in the latter there should be enlargement of the left ventricle in addition to the dilated left auricle. More important for the recognition of mitral stenosis are dilatation and hypertrophy of the right ventricle, which occur secondary to increased pressure in the pulmonary circulation. Stasis in the pulmonary circulation with dilatation of the pulmonary vessels is also a confirmatory sign. The dilated left auricle may not be visible in the direct posteroanterior view, but the left border of the heart is usually straighter than normal, often actually convex, in part due to enlargement of the right ventricle and its outflow tract, the conus pulmonalis. In most cases, both the enlarged left auricular curve and the enlarged conus pulmonalis above it are visible on posteroanterior or anteroposterior roentgenograms and at roentgenoscopy. The *right* anterior oblique position is particularly valuable in demonstrating enlargement of the left auricle posteriorly, and barium may show the characteristic concave indentation of the esophagus in this position. This extends from a point below the indentations of aorta and right bronchus to a point 2 or 3 cm. above the diaphragm. The *left* anterior oblique view is valuable for enlargement of the right ventricle, which will project anteriorly in this view, forming more than the normal one-third of the heart shadow as determined by a perpendicular line through the center of the ascending aorta. Another associated finding which is present when the mitral valve involvement was acquired in childhood is hypoplasia of the aorta, with a small or

indistinct aortic arch. Dilatation of the conus pulmonalis may help to obscure the arch of the aorta, but more often the small arch is real rather than apparent. With advanced disease and marked enlargement of the left auricle, its right border may form a second convex curve on the right border of the heart above the curve of the right auricle. A difference in pulsation of the two curves is readily apparent under the fluoroscope, the right auricle contracting first, followed by an expansion of the left auricle, presumably due to regurgitation with ventricular systole. A dilated pulsating descending branch of the right pulmonary artery may simulate this condition, but with experience the two can be differentiated. The left bronchus may be elevated and even compressed by such aneurysmal dilatation of the left auricle but this is of little value in differential diagnosis as it occurs only with advanced disease. Its demonstration, however, may explain a troublesome cough or dyspnea. Calcification in the diseased mitral valve is relatively frequent and is helpful in both anatomical and etiological diagnosis. It will be discussed separately.

Fig. 1. Mitral stenosis, compensated. H. R., female, aged thirty-four; chorea at age of nineteen. Roentgen examination for cough and dyspnea revealed slight cardiac enlargement, straight left border, dilated left auricle posteriorly. Dr. Levine found definite short presystolic murmur, confirmed diagnosis. Decompensation first occurred in February, 1938: dyspnea, cough, enlarged liver and edema. Patient died July 26, 1938. No calcification of valve.

It has been said (Parkinson)[3] that definite dilatation of the left auricle is one of the most certain signs of mitral stenosis, second only to the characteristic late diastolic or presystolic rumbling murmur. It is important, therefore, to know what other conditions may cause enlargement of the left auricle and what sources of error must be considered. In my experience, heart failure without mitral valve disease is the most confusing. Here there may be actual dilatation of the left auricle due to stasis as well as general enlargement of the heart. Fortunately we rarely see patients with this degree of failure as they are too sick for roentgenoscopy, but the findings in bedside or portable films may be indistinguishable from mitral disease. It may well be said here parenthetically that we consider a careful roentgenoscopic examination much more important and valuable than any number of films, the latter serving chiefly for records and for measurement of size. Complete heart block and fibrillation of the auricles are the next most common sources of roentgenographic error. We hesitate to make the diagnosis of organic disease of the mitral valve in the presence of complete heart block or of auricular fibrillation unless calcification of the mitral valve can be demonstrated. Sussman and Woodruff,[9] however, have shown that actual dilatation of the left auricle is rare in auricular fibrillation unless complicated by valvular disease. Congenital heart disease, particularly patent ductus arteriosus, pulmonic stenosis or interauricular septum defect are possible sources of error, but they are relatively quite rare and are usually readily distinguishable from mitral disease. The converse is more often true. The pulmonary conus may be so dilated in mitral stenosis that

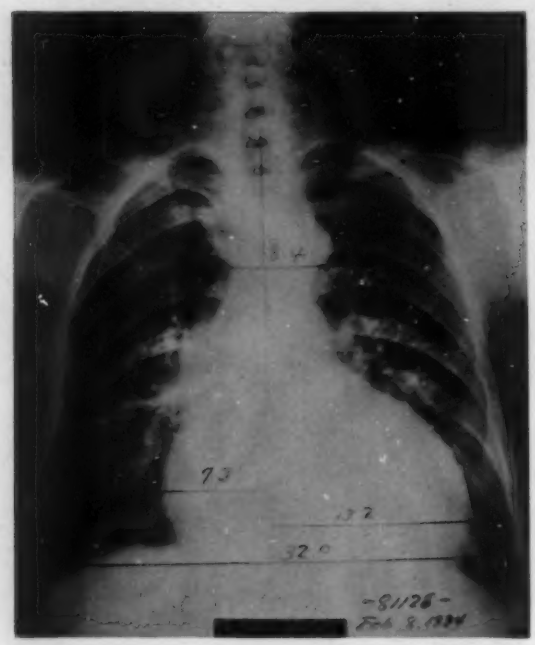


Fig. 2. Aortic stenosis, acquired, rheumatic. G. H., male, aged sixty-six. Chief complaint dyspnea, slight for years, worse in past few months. No history of rheumatic fever or syphilis. Wassermann reaction negative; blood pressure 145/96. Gallop rhythm, grade 2 basal systolic and faint aortic diastolic murmurs. No thrill. Roentgen examination for evidence of luetic aortitis or aortic stenosis revealed typical "aortic heart," calcification in aortic valve. Improved under cardiac therapy and aortic systolic thrill appeared. Died April 5, 1934.

the error may be made of considering the case one of congenital heart disease, overlooking the posterior enlargement of the left auricle. Hyperthyroidism is often given as a cause of error, but in my experience this rarely simulates mitral valve disease, and then only when heart failure or auricular fibrillation or both are present.

AORTIC VALVE

Aortic insufficiency without aortic stenosis may be either rheumatic, syphilitic or hypertensive in etiology. The first is much more common in New England. A few cases in old people are probably due to atherosclerotic changes involving the aortic valves. In young persons it is nearly always the result of rheumatic involvement; in middle age it may be rheumatic or syphilitic; in old people it is usually rheumatic or hypertensive.

Clinically aortic insufficiency is diagnosed correctly in almost 100 per cent of the cases so that roentgenological assistance is rarely necessary. When seen, the striking changes apparent are the excessive enlargement of the left ventricle and the exaggerated alternating pulsation of aorta and left ventricle, giving the characteristic "rocking beat." When due to syphilis there may be an associated dilatation of the ascending aorta.

The characteristics roentgenologically of aortic insufficiency, therefore, are marked enlargement of the left ventricle usually without enlargement of the other chambers until failure begins, and the vigorous rocking beat corresponding well to the water hammer or Corrigan pulse. The enlargement may be downward and to the left in an oblique direction rather than more transverse as in aortic stenosis. The aortic knob is usually more prominent than normal. A beat simulating that of aortic insufficiency may be found in patients with a left-sided pneumothorax, with hyperthyroidism and occasionally in marked anemia. The differentiation is usually not difficult.

Aortic stenosis is usually the result of rheumatic fever, is more common in males, by a ratio of 3 to 1, and is compatible with a long and healthy life, which probably accounts for the fact that so many of the cases are first discovered in older persons. At our hospital we feel that the majority of cases are the result of rheumatic infection and that atherosclerosis (Mönckeberg's sclerosis) is relatively rare. This is based on the fact that 50 per cent of these patients give a history of preceding rheumatic infection, a frequency nearly equal to that in chronic mitral valvular disease. Furthermore, many of these patients are old enough to have forgotten apparently unimportant childhood diseases, their memory may be faulty, and their parents (from whom most of the histories of rheu-

matic fever or chorea are obtained in younger patients) are usually inaccessible or dead. Unlike mitral valvular disease, however, when failure does set in the prognosis changes rapidly from good to poor, and death usually soon follows. Clinically there is a larger percentage of error than in the other groups so far discussed. In general practice the diagnosis is made in only 40 per cent of the cases, and by expert cardiologists in 53 per cent, as stated by Thompson and Levine. Here the roentgen examination should be of considerable value, but the roentgenological appearances are not characteristic. There is moderate enlargement of the left ventricle, usually more rounded and blunt than in aortic regurgitation and more transverse; less prominence of the aorta, both to the right and at the arch; and a beat with smaller amplitude and more sustained than in insufficiency. Actually there is more hypertrophy and less dilatation than in aortic regurgitation and, if of equal size, the heart with aortic stenosis will weigh more than that with regurgitation alone. The great majority, if not all, of hearts with primary aortic stenosis show at autopsy large calcium deposits in and around the aortic valves, and most of these are demonstrable roentgenoscopically. This is by far the most important and valuable finding in differentiating aortic stenosis from other conditions causing left ventricular hypertrophy, and particularly from syphilitic aortic valve disease. We have not yet found calcium deposits visible roentgenoscopically in uncomplicated syphilitic aortic valve disease.

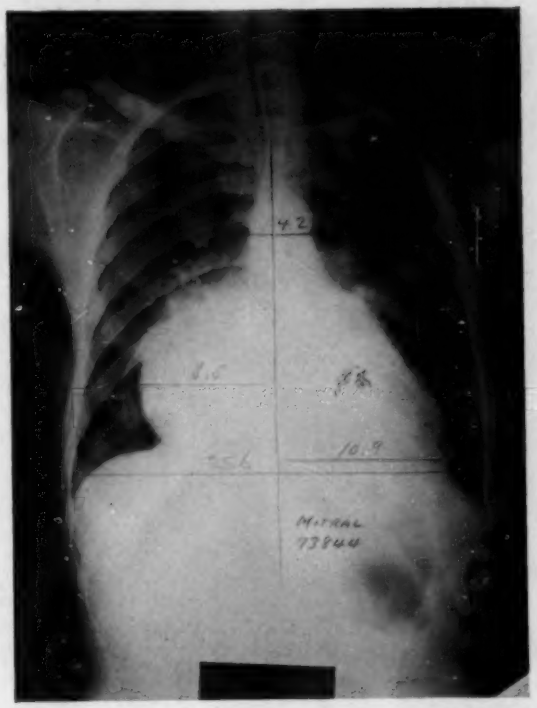


Fig. 3. Combined mitral and aortic valvular heart disease, acquired, rheumatic, and acquired relative tricuspid insufficiency. S. A. J., male, aged forty. Chief complaint cough, hemoptysis, dyspnea, orthopnea, edema. Rheumatic fever at age of ten. Ten admissions to Peter Bent Brigham Hospital, 1923 to 1937, for cardiac decompensation. Systolic and diastolic murmurs at apex, diastolic murmur at base in aortic area. Blood pressure 108/68. Gradual enlargement of liver, ascites and edema. Died August 28, 1938. Calcification noted roentgenoscopically in mitral valve area (dotted area in illustration) in 1932. Most of the heart shadow is made up of dilated auricles; marked dilatation of right auricle.

COMBINED VALVULAR DISEASE

The most common combination in long standing acquired rheumatic valvular lesions is aortic and mitral involvement, and this combination is more frequently seen than either valve lesion alone, greater even than the sum total of the single valve lesions, according to some authors, particularly those using autopsy statistics. The accuracy of clinical diagnosis in combined lesions varies, but in general is the same as noted above for mitral disease, better for aortic stenosis, but less accurate for aortic insufficiency when mitral disease is also present. We can disregard the terms "organic" and "relative" insufficiency as we are dealing with cardiac contours, and the change in the relative size of the different chambers is apt to be the same whether the regurgitation is due to structural changes in the valves or to a physiological change in the ring supporting the valve. The time factor or the duration of the stenosis or regurgitation is more im-

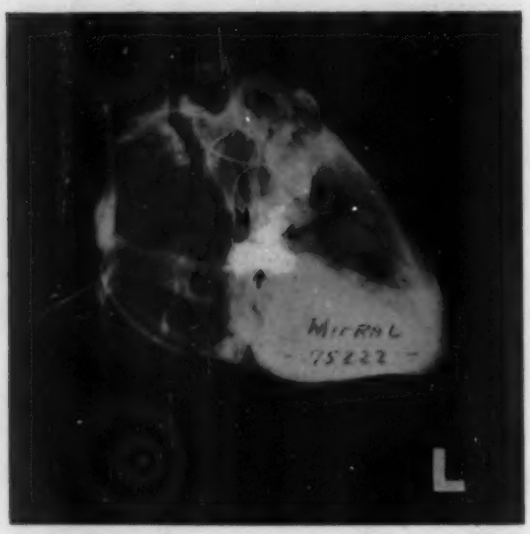


Fig. 4. Calcified mitral valve, post-mortem roentgenogram. J. E., male, aged forty-four. Rheumatic fever at age of twenty-two; known heart disease since. Chief complaint dyspnea, precordial pain, occasional hemoptysis. Physical examination showed cyanosis, slight cardiac enlargement; blood pressure, 118/70. Apical diastolic murmur and thrill, auricular fibrillation, pulmonary congestion. Calcification found roentgenoscopically in mitral valve on October 18, 1932. Died November 8, 1932. Autopsy. Heart weighed 620 gm. Fishmouth stenosis of mitral valve with calcified mass in posterior leaf.

portant, as well as the degree of involvement. As one valve is usually involved first and the other one some time later, we have variable contours or shapes depending on the sequence. Thus there may be marked left ventricular enlargement with relatively moderate dilatation of the left auricle, suggesting first or primary involvement of the aortic valve and secondary or subsequent mitral valve lesion, either organic or "relative." As previously stated, calcification of only one valve in such a case would suggest that it was the seat of the first or original disease. Similarly, according to the physical signs and symptoms, an interpretation of "predominant" and "secondary" valvular disease may be made.

Roentgenologically we find cardiac enlargement in all cases of combined valvular disease, and the particular dilatation or hypertrophy depends upon the major or predominant lesion, modified by secondary changes in the intracardiac pressures. Thus in aortic and mitral valve disease with primary aortic stenosis and secondary mitral involvement, there is marked enlargement of the left ventricle, which may obscure the dilated left auricle in the posteroanterior view, but the latter will be demonstrable in the right anterior oblique view. There will be little or no prominence of the conus pulmonalis, the aortic knob will be prominent, and the calcified areas, if present, will be found in the aortic valve area. Similarly, a primary or predominant mitral valve disease with secondary involvement of the aortic valve, either organic or relative, will show predominantly the characteristics of mitral valve disease —marked dilatation of the left auricle, prominent conus pulmonalis, small aortic knob and enlarged right ventricle, modified by more left ventricular enlargement than usual, a pulsation greater in amplitude than in mitral disease alone, and the calcification, if present, will be in the mitral valve region. Less frequently both valves will become involved at an early age, in which case the cardiac silhouette is a combination of the two types of contour equally and both valves may be heavily calcified. The aortic valve lesions are more common alone than mitral valve lesions alone. Primary involvement of the mitral valve with secondary involvement of the aortic valve is said to be three times as frequent as primary aortic involvement with secondary mitral valvular disease (White).[13] Strangely enough the prognosis as to length of life is better with combined valvular disease than with single lesions, except aortic stenosis alone.

TRICUSPID VALVE

In many of the cases of combined valvular disease the tricuspid valve becomes involved, usually late in the course of the disease. We have no case of tricuspid valve disease alone; it is always associated with disease of the mitral or aortic valve or

both. Autopsy figures given by Wauchope[12] in 416 cases of rheumatic valvular disease show the tricuspid valve involved in 130, or 31 per cent. The involvement is nearly always a relative regurgitation, with the well known clinical signs of pulsating veins in the neck and pulsating enlarged liver. The development of tricuspid regurgitation may be followed by apparent clinical improvement, and roentgenologically there is seen decrease in the amount of pulmonary congestion as the probable cause. Roentgenoscopy and the roentgenograms will also show marked enlargement of the right auricle and right ventricle, and the lower right cardiac contour may reach the right axillary border of the chest. The lung fields may be remarkably clear and the superior vena cava may show a marked systolic pulsation. Occasionally the liver pulsation is visible roentgenoscopically as transmitted to the right diaphragm.

Possible sources of error are hiatus herniae of the fundus of the stomach projecting to the right and sometimes showing transmitted pulsations, and a tortuous descending aorta which occasionally projects to the right of the lower cardiac contour. Marked scoliosis may confuse or obscure right-sided cardiac enlargement, but is rarely erroneously interpreted.

Rarely tricuspid stenosis occurs associated with aortic or mitral valvular disease and is probably due to the same rheumatic infection. This condition may give peripheral signs simulating adhesive constrictive pericarditis, as discussed by Dr. Freedman. The differentiation is easily made by demonstrating marked cardiac enlargement, particularly of the right auricle. In adhesive constrictive pericarditis, the heart is nearly always normal in size or even small.

The clinical diagnosis of tricuspid valvular disease is quite inaccurate, as shown by autopsy statistics, but the error is of relatively little importance as the major concern is with the associated mitral or aortic valvular disease, and the general picture of myocardial failure. In the rare cases of tricuspid stenosis, however, the prognosis may be much better than with the same evidence of cardiac failure without tricuspid stenosis.[11]

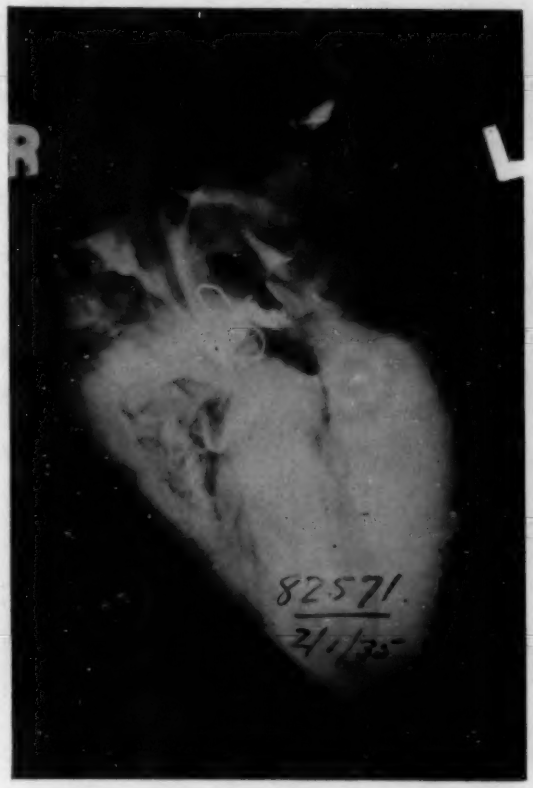


Fig. 5. Calcified aortic valve, post-mortem roentgenogram. Note persistence of shape of aortic cusps, pointing upward into aorta. Slight calcification of margin of mitral valve below and to right of aortic calcification. A. R., female, aged seventy-two. No history of rheumatic fever. Quite well until May, 1933, when first decompensation occurred following exertion. Never fully recovered; died in January, 1935. Physical examination showed rough high-pitched systolic murmur all over precordium, maximum at base. Questionable systolic thrill at base became definite after roentgenoscopic demonstration of calcification in aortic valve. Autopsy. Heart weighed 420 gm. Marked stenosis of aortic valve, rheumatic type. Slight stenosis and calcification of mitral valve. Healed endocarditis of tricuspid valve without stenosis or insufficiency.

PULMONARY VALVE

Lesions of the pulmonary valve are practically always congenital in origin and will be discussed by Dr. Roesler. When the pulmonary valve is affected by rheumatic

Fig. 6. Calcification of mitral annulus fibrosus. Originally (December, 1932) interpreted as calcification in mitral valve. P. M., male, aged seventy-six (December, 1932). Chief complaint angina since 1930. No history of rheumatic fever. Rough systolic murmur heard all over precordium. No thrill. Blood pressure 120/65. Heart not enlarged. Wassermann reaction negative. Clinically coronary disease, myocarditis,? aortic stenosis and insufficiency. Last seen April, 1938, recovering from coronary infarct (aged eighty-two). Roentgenogram taken in left anterior oblique position, almost lateral, shows typical U-shaped mass of calcification in posterior half of heart shadow.

fever there is no resultant deformity as in the other valves. Relative insufficiency may occur, in which case there is enlargement of the right ventricle, best demonstrated in the left anterior oblique view of at least 60 degrees. Any disease or condition resulting in increased intrapulmonary pressure may cause similar hypertrophy of the right ventricle—emphysema, pulmonary fibrosis, pulmonary endarteritis, etc.

CALCIFICATION OF THE VALVES

Almost all of the above statements are standard facts or beliefs and available in many of the textbooks on cardiology or roentgenology. My particular desire in this paper is to stress the frequency of calcification of the diseased valves, the methods and procedures in demonstrating them and the importance of this additional finding.

Calcium deposits occur in the great majority of chronically diseased valves, as can be demonstrated by roentgenograms of the excised heart at necropsy. Frequently these deposits are large enough and sufficiently opaque to be demonstrable by roentgenoscopy. To search for them requires only a few seconds in routine roentgenoscopy, and the one and only important rule is to look *through* the heart rather than at its contours, watching for dark irregular shadows which have a characteristic dancing motion between cardiac systole and diastole. The movement is usually along an oblique line between the apex and base of the heart but may be triangular rather than linear.

An interesting chapter might be written on the movements of the valves, but in brief we may say that in most cases the aortic valve moves toward the apex with systole, starting its motion synchronously with the first sound, ending at the second sound and returning during diastole to its original position. The mitral valve frequently but not always shows a short excursion toward the base of the heart (cephalad) at or with the first sound, then toward the apex during the remainder of systole, returning during diastole. Both may show an angular movement during systole, first toward the diaphragm (caudad) and then toward the apex of the heart, returning in a straight line during diastole. To observe and time these movements we have listened with a stethoscope at the apex while watching the movements of the valves, counting out loud either "Lub-dup" or better "one-two, one-two." Sometimes we have had a cardiologically-minded colleague listen and count while we observed. The timing is fairly easy in slow regular hearts, difficult or impossible in rapid or irregular hearts. Artificial methods

of slowing the heart would be advantageous but have not yet been tried.

When both the aortic and mitral valves are calcified, there may be a perceptible difference in their movements, the aortic preceding the mitral by a fraction of a second, and sometimes producing a slight see-saw or teeter-totter appearance.

The amplitude of movement is striking, usually exceeding that of any visible part of the cardiac contour and probably indicates the marked pumping action played by the valves, the base of the heart, and the auriculoventricular "septum." As a rule, the valve moves a greater distance toward the apex of the heart than the apex does toward the base. We have often recorded an amplitude of excursion of 2.5 to 3.0 cm. between systole and diastole.

To see these shadows easily one must have his eyes adequately prepared, with at least fifteen minutes in a completely darkened room before roentgenoscopy. We routinely search for intracardiac calcification in all patients (*a*) who are suspected of having heart disease, (*b*) in whom heart disease is evident, (*c*) with enlarged hearts, (*d*) who are over sixty years of age. No special apparatus is required, but we find that a small fluoroscopic aperture and a screen of high intensity, such as the type B, give best results. We use a 5 inch gap and 5 ma. with the above conditions. To locate the calcified valves, we rotate the patient into a slight right anterior oblique position, about 15 to 20 degrees, then look *through* the heart along the line of the auriculoventricular sulcus. This can be identified by finding the auriculoventricular junction on the left border, and searching obliquely across the heart shadow downward and medially 45 degrees from the horizontal from this point. In this position both the aortic and mitral valvular calcification is best seen, but difficult to differentiate. Having found intracardiac calcification, we rotate the patient to the left anterior oblique position for differentiation. In this position the mitral valve is in the posterior third of the heart shadow while the aortic valve is in the middle third. Confusing shadows such as calcified glands, costal cartilages, Ghon tubercles, etc., are easily ruled out by their lack of motion with systole and diastole. On roentgenograms these confusing shadows are more difficult and sometimes impossible to differentiate. The roentgenograms must be made in one-twentieth of a second or less to be satisfactory. Both at roentgenoscopy and roentgenography it is advantageous to have the patient take a deep inspiration and hold it as this facilitates search by stopping respiratory movements and adds contrast by distending the overlying lung.

The calcified valves will show up as dense, irregular, nodular masses similar in appearance to calcified mesenteric glands on the film, and of course as darker shadows within the dark heart shadow on the screen. We have frequently found a denser, more solid mass of calcium in the annulus fibrosus of the mitral valve in aged patients. This is always more homogeneous, and is J-shaped or U-shaped in appearance. It is a senile change entirely and usually exists without demonstrable heart disease, although it may be associated with other lesions. It is frequently associated with arcus senilis and with calcification of the intervertebral discs, also senile changes. Its movements are quite similar to those of the mitral valve.

The frequency of demonstrable intracardiac calcification is astonishing. Since 1931, when, at the suggestion of Dr. Christian[2] we began looking for intracardiac calcification, we have found almost 200 patients in our small clinic with definite and roentgenoscopically demonstrable intracardiac calcium deposits, exclusive of calcification in the walls of the aorta. The majority of these are in the aortic or mitral valves, but in looking for calcified valves we have found and demonstrated heavy calcium deposits in the coronary arteries, in the annulus fibrosus, in the pericardium, and even in the myocardium. Our figures and percentages are not accurate as to relative frequency, as many cases of known

chronic valvular disease seen in the clinic are not referred to us but our record at present reads:

Calcification in aortic valve	59 cases
Calcification in mitral valve	56 cases
Calcification in coronary artery	35 cases
Calcification in annulus fibrosus	22 cases
Total	172 cases

Twenty of these have come to autopsy and in every case massive deposits of calcium have been found. There was one error in roentgenological interpretation in this group, a deposit of calcium in the aortic valve being interpreted as calcification in the coronary arteries. This is a positive error of only 5 per cent. The negative error is much greater, as we have seen many cases of chronic valvular heart disease at autopsy with small or moderate-sized calcium deposits in the injured valves, which had been overlooked or missed at roentgenoscopy.

We believe that the demonstration of calcification in one or more valves is a definite help not only in the diagnosis of organic or structural disease but also in etiological diagnosis, as the great majority (excluding, of course, the annulus fibrosus) are probably consequent to the damage done years before by rheumatic fever. As previously stated, this is particularly valuable in differentiating luetic from rheumatic aortic stenosis.

SUMMARY

1. Acquired valvular heart disease results in cardiac enlargement in almost every case and causes typical changes in the cardiac contour in the great majority.

2. Enlargement of the left auricle is one of the most reliable signs of mitral valvular disease.

3. The contour of the heart in aortic valvular disease may be duplicated by hypertensive heart disease but the differentiation can frequently be made by roentgenoscopy—in aortic regurgitation by the "rocking beat" and in aortic stenosis by demonstrating calcification in the aortic valve.

4. Calcification in aortic and mitral valves is frequently demonstrable roentgenoscopically and adds considerably to the accuracy in both structural and etiological diagnoses.

REFERENCES

1. Cabot, R. C. Facts on the Heart. W. B. Saunders Co., Philadelphia, 1926.
2. Christian, H. A. Aortic stenosis with calcification of the cusps. *J. Am. M. Ass.*, 1931, *97*, 158–161.
3. Parkinson, J. Enlargement of the heart (Lumleian Lecture). *Lancet*, 1936, *1*, 1337; 1391.
4. Parrot. Quoted in Osler, W.: Principles and Practice of Medicine. Thirteenth edition. Revised by H. A. Christian. D. Appleton-Century Co., New York, 1938, p. 1007.
5. Roesler, H. Clinical Roentgenology of the Cardiovascular System. Charles C. Thomas, Springfield, Ill., 1937.
6. Sperling, P. Ueber Embolien bei Endocarditis. Inaug. Diss., Berlin, 1872.
7. Sosman, M. C., and Wosika, P. H. Calcification in aortic and mitral valves. Am. J. Roentgenol. & Rad. Therapy, 1933, *30*, 328–348.
8. Sosman, M. C., and Wosika, P. H. Position of heart valves and their relation to anterior chest wall in living subjects with abnormal hearts. *Am. Heart J.*, 1934, *10*, 156–162.
9. Sussman, M. L., and Woodruff, M. T. Significance of left auricular dilatation in auricular fibrillation. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, 184–188.
10. Thompson, W. P., and Levine, S. A. Comparison of accuracy in diagnosis of single and multiple valvular disease of the heart. *New England J. Med.*, 1936, *215*, 670–671.
11. Thompson, W. P., and Levine, S. A. Note on duration of symptoms and age at death in chronic rheumatic valvular disease, especially in tricuspid stenosis. *Am. J. M. Sc.*, 1937, *193*, 4–7.
12. Wauchope, G. M. Clinical importance of variations in number of cusps forming aortic and pulmonary valves. *Quart. J. Med.*, 1928, *21*, 383–399.
13. White, P. D. Heart Disease. Second edition. Macmillan Co., New York, 1937.

DISCUSSION

Dr. George W. Holmes, Boston. The important part of Dr. Sosman's paper, as he said, is the demonstration of calcification in the valves. He deserves great credit for this work, which has given us a new diagnostic sign. It not only helps in determining the presence of

disease but it gives some idea as to the etiology of the disease.

I would like to emphasize a point which he brought up, and that is the necessity of careful work, of taking time to get your eyes properly dilated before you attempt to look for this sign. You will miss them all if you attempt to do the work intermittently during your regular office work. Most of us cannot take fifteen minutes between each patient to get our eyes dilated. You must have these patients in a group or along with another group in which you are making fluoroscopic examinations and, as he has said, it takes at least fifteen minutes. If you cannot do that, it is useless to attempt the examination.

Then, it helps a great deal if you can see a case with a man who is well trained and who has already made that diagnosis. We find that our residents, for instance, will miss this thing right along until one of the older men takes them in and points out the shadow. It would seem that anything as easy to see as this, after it has been pointed out to you, would be difficult to miss, but that does not seem to be the fact. One usually has to have the shadows pointed out to him for the first time. After that, you wonder why you haven't seen them before. Then, too, as Dr. Sosman has said, the condition is quite common and we should be on the lookout for it. I think it is a very important contribution to the roentgen diagnosis of cardiac disease.

Dr. Samuel Levine, Boston. I have been very much interested in auscultation; it has been very valuable in the diagnosis of valvular diseases, notwithstanding the fact that there have been teachers in the recent past who told us to throw the stethoscope away. I think we might just as well throw it away if we are thinking in terms of the early diagnosis of tuberculosis and go straight to the roentgenologist. But valvular disease is the clinician's forte. We can do quite well even without the roentgenologist, not quite as well as Dr. Sosman's figures indicate. We published some time ago figures of the accuracy of diagnosis of various valve diseases, mitral stenosis, aortic stenosis, combined lesion, tricuspid valve disease, and compared the accuracy of the general medical service, the house officers, the visiting men, with the so-called cardiac experts, the group of us who work in the cardiac clinic, and we did top the list. But that has reference to advanced disease. We can do very well in the diagnosis of valve disease when it is well advanced. I do not believe that similar studies on cases ten or twenty years before they die would show such an excellent correlation. At that stage, we do need all the help we can get. There is no such study available at the present time as to how accurate we are clinically in the diagnosis of early mitral stenosis, how we would do without the roentgen ray and how much better we would be with it or how good the roentgen ray would be without us. How many of these prominent left auricles in the early stage of disease really mean mitral valve disease? I suspect some of them do. I feel at the present moment fairly certain that they do not all mean mitral valve disease, but I do not know. We do not have the opportunity of checking that, because they live; they die twenty years later. It is going to be a difficult task, but it must be done sometime, to check up the accuracy of the roentgen findings in comparison with the clinical findings in the early stages of valvular disease. There are some early diagnoses that we can make. We can make the diagnosis of aortic insufficiency if we hear a basal diastolic murmur.

The pathologist cannot tell us what the dynamics of that aortic valve were when the heart was alive. The tone is gone, the pressure is gone, the aortic ring may have a little different caliber, the valves may be normal in appearance, but in life they may not have closed the gap and there may have been some regurgitation. So I do not feel chagrined one bit if I definitely hear the sign of aortic insufficiency in life and find the valves are normal or apparently normal pathologically. That occurs in hypertensive people with normal valves, with a pressure of 240 mm. The aortic ring and the aorta itself may have been dilated, resulting in some insufficiency which is present in life, which manifests itself by an aortic diastolic murmur, yet valvulitis is not apparent.

Therefore, figures of the sort that we heard from Dr. Sosman, and for which I am partly responsible, may be misleading because they are the easy cases to diagnose. We can diagnose mitral stenosis with a high degree of accuracy when it is full blown, yet occasionally we fumble entirely. There was one case in which I could not hear any evidence of mitral stenosis. There are some advanced cases which are terribly sick, in whom the dynamics may be so sluggish that the rumble on which we mainly base our diagnosis of mitral stenosis is absent.

With the most careful auscultation, I could hear no diastolic murmur, yet it was an outspoken case of mitral stenosis. With careful auscultation, in the great majority of cases, we are able to hear the diastolic murmur of mitral stenosis, but once in a while we get invaluable aid from the roentgen studies.

The studies on calcification of the valves have helped a great deal in the diagnosis of aortic stenosis. The accuracy of diagnosis used to be poor. It is a great deal better now, and it particularly helps the clinician in distinguishing what he might otherwise suspect to be a luetic aortitis. In both conditions there is a systolic murmur at the base and nothing else. You need not even have cardiac failure either in aortic stenosis or luetic aortitis, and we know that once in a great while, even cases of luetic aortitis have a negative Wassermann, so that we may need all the help we can get from the roentgenologist. When we can find calcified aortic stenosis, that rules out lues. We have never seen a case of luetic disease of the aorta or aortic valve result in a constricting stenosis. It destroys the valve and produces regurgitation, but it does not produce aortic stenosis. The finding of calcification stamps the case, therefore, as either rheumatic, which we believe is generally the case, or so-called Mönckeberg's arteriosclerosis. Only last week, I had that very experience. I saw a patient in my office, and my first diagnosis was luetic aortitis. Dr. Sosman examined the patient fluoroscopically and found definite aortic stenosis. That not only helps in the anatomic diagnosis, but in the etiologic diagnosis.

Dr. Sosman mentioned my diagnosing rheumatic valvular disease from the patient's freckles. I think he is beginning to believe that there is something to it. There are certain people who are rheumatic. We have no test for rheumatic fever. The classical case we all know, but these vague, atypical cases you can diagnose only by getting this clue or that clue. Among these are nosebleeds, undue freckling, illnesses that came in childhood for no reason, repeating themselves each March, and so on. There is no chemical or bacteriological test for rheumatic fever and only half of the patients who have bona fide rheumatic fever have a history of it. The others have had it but can give no history of it. This nurse to whom Dr. Sosman referred (Fig. 1) was the typical rheumatic type. She would also have been one of the cases in which the roentgenologist finds evidence of mitral stenosis where the clinician does not, although it would depend upon which clinician it was. I had no difficulty in finding the rrup-rrup-rrup or the short rumble of the mitral stenosis whereas it was missed and she was regarded as having nothing wrong with her heart, by others. I do not mean this as boastful, but you can see statistics may be vitiated. The roentgenologist was helpful to the fellow who was not well trained, but was not helpful to me one bit. I knew she had mitral stenosis and a good many well trained internists would have known it.

I am going to make one comment about the prognosis of the various valve diseases. In aortic stenosis the patient remains well for a long time. He is in good health; he may have aortic stenosis, with calcification, but he will have a regular heart, and feel fit for a long time. But when he fails, he fails rapidly. On the average, he will have the longest life and the shortest illness after failure. The mitral stenosis patient dies about ten years younger, but he has had disability for a longer time. Once they fail, you expect them to get better, then they fail again and improve. However, the age at death is a good deal less than the age at death of the aortic stenosis patient. Curiously enough, the tricuspid patient does even better than the mitral stenosis patient. He lives longer after symptoms develop. All cases of tricuspid stenosis that we have seen have had mitral stenosis and some have had aortic stenosis, too. Once their disability starts, they live a long time, three or four years longer on the average than the mitral case, and the reason for it is that they have the equivalent of a mechanical pericardial constriction inside the heart rather than on the outside. They have symptoms from a mechanical impairment, not from heart muscle failure necessarily.

The blood cannot get into the heart because of a constriction inside the right ventricle in contrast to the pericardiac constriction that occurs in non-valvular cases, outside the heart. Now, a mechanical process does not indicate necessarily any myocardial failure. The blood just cannot get in, so that is a more static situation. The result is that the average span of life of tricuspid cases has been seven years or more after failure starts. In the aortic case, it is about a year or two and in the mitral case, about three or four years. It is a rather curious but an apparently anomalous situation.

One word also about aortic cases in contrast

to mitral cases from a roentgenologic point of view. The aortic case has a smaller silhouette than the mitral, but, what is a good deal more important, the average weight of the aortic stenosis case is 100 to 200 grams greater than in those with mitral stenosis, although the silhouette is smaller. There is a good deal more dilatation in mitral cases which have these large right and left auricles containing 250 to 500 cc. of blood in each chamber or sometimes a liter of blood in each auricle. This tremendous dilatation results in a thin-walled structure, whereas in aortic stenosis there is not so much dilatation but there is a huge thickening of the ventricle resulting in a 500 or 900 or 1,000 gram heart.

One thing I learned about the heart from the calcification studies is the surprising place where these valves are located. We speak about the aortic area being in the second right interspace. That is what we teach our students. But the aortic area is almost down near the epigastrium, close to the fourth or fifth space. That first came to my attention when I saw where these calcified valves were located. That also explains why we may feel a thrill in that area. It is not infrequently felt in the mid-precordium rather than the aortic area. The flow of blood is in that direction and it is generally felt best at the base, but it is often felt over the mid-precordium as well as over the so-called aortic area.

In general, I would like to emphasize that the early diagnosis of mitral stenosis can almost always be made by careful auscultation. Have the patient lie on the left side or have him hop around for twenty hops and then listen for that first half-minute after the exercise for that short rumble. It will come out for ten or fifteen seconds and then it dies away but it already tells the story that there is early constriction.

It may be that the prominent left auricle may be seen at an earlier stage; that I do not know. We hope to have an answer some day. Sosman and I are making notes on some of these early cases, seeing which one is going to be right. We may have something to say later on, but at the present time I do not know how to interpret the slightly prominent left auricle when I can't hear the clinical signs of mitral stenosis in a person who is rheumatic.

ROENTGENOLOGIC ASPECTS OF NON-VALVULAR DISEASE OF THE HEART*

By GEORGE LEVENE, M.D.
BOSTON, MASSACHUSETTS

CHANGES in cardiac size, contour, and activity are not infrequently encountered in systemic disease. While, in many cases, these alterations can merely suggest the presence of an abnormal state, the nature of which can be determined only by supplementary clinical and laboratory examinations, in others they are sufficiently distinctive to identify the underlying cause.

FIG. 1. Left ventricular heart failure (congestive failure) in a case of chronic hypertension. Hypertrophy and dilatation of the left ventricle indicate hypertension. Note congestive infiltration of the lungs resulting from insufficiency of the left ventricular myocardium. Dyspnea is a prominent symptom.

The contour of the normal heart is maintained as a balance of two factors: the amount of muscle and its tone; and it is responsive to variations in these factors resulting from local or constitutional disease. Variations in the amount of muscle are proportionate to the amount of work to be done, and the changes are localized only to the chamber involved in the demand for increased or diminished activity. This is well illustrated by the pure hypertrophy of the right ventricle in stenosis of the pulmonary artery, or of the left ventricle in stenosis of the aortic valve, and by the muscular atrophy of the left ventricle in stenosis of the mitral valve. Changes in tone are chiefly dependent upon the nutrition of the myocardium resulting from local or constitutional damage.

MYOCARDIAL INSUFFICIENCY

The acute forms of this condition are produced by infections, poisons or sudden severe strain in a heart with diminished reserve. From the standpoint of cardiac dynamics, the result, in each case, is the same. The tone of the heart muscle is impaired, so that the muscle fibers relax. If the cause is systemic, the entire heart is affected, with consequent enlargement of all the chambers. There is diminished amplitude of cardiac contractions with congestive infiltration of the lungs. Cardiac asthma is a form of acute myocardial insufficiency, characterized by marked dyspnea. While it is usually possible to recognize the existence of myocardial damage, the appearances are not always characteristic of distinctive lesions.

The more chronic forms of myocardial insufficiency may involve either or both sides of the heart, the commonest form being left-sided failure.[12] These cases show a moderate increase in transverse diameter (evidence of relative mitral insufficiency),

* From the Department of Radiology, Massachusetts Memorial Hospitals, Boston. Read in a Symposium on Heart Disease at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938.

superimposed upon the antecedent cardiopathy, which, most often, is hypertension, coronary thrombosis or aortic valvular disease. There is congestive infiltration of the lungs (Fig. 1), and, not infrequently, an interlobar or pleural effusion. Dyspnea is the most prominent symptom, the degree being more or less parallel to the amount of myocardial damage. A good index to the integrity of the heart muscle is therefore obtained by noting the difficulty the patient experiences in holding his breath during roentgenoscopic examination.

Right-sided failure usually follows changes on the left, though it may exist as a single or primary lesion. The tricuspid valve becomes incompetent so that the pulmonary congestion noted in left-sided failure is largely relieved (Fig. 2). The lung fields become clearer and dyspnea is markedly improved. There is a variable degree of edema and ascites with engorgement of the liver and veins of the neck. The heart shows a greater increase in transverse diameter, due principally to dilatation of the right ventricle. There is a loss of convexity of the borders of the heart, more marked on the left, the change being parallel to the degree of impairment and in inverse ratio to the amount of previous hypertrophy.[5] Roentgenoscopically, the amplitude of contractions is diminished and systolic pulsations are observed in the superior vena cava. This, in general, is the appearance of the heart in myocardial impairment. Certain of the non-valvular cardiopathies, however, produce more or less distinctive changes.

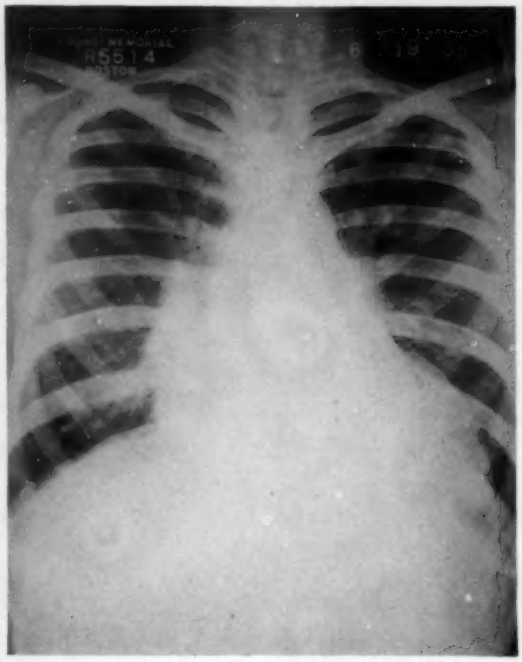


Fig. 2. Right ventricular failure (frequently preceded by changes on the left side), from a case of hypertensive heart disease. Note enlarged right chambers and comparatively clear lung fields. In these cases, congestive infiltration of the lungs noted in left ventricular failure (Fig. 1) is relieved, dyspnea is improved, but edema and ascites become prominent symptoms. Systolic pulsations of the superior vena cava are observed roentgenoscopically.

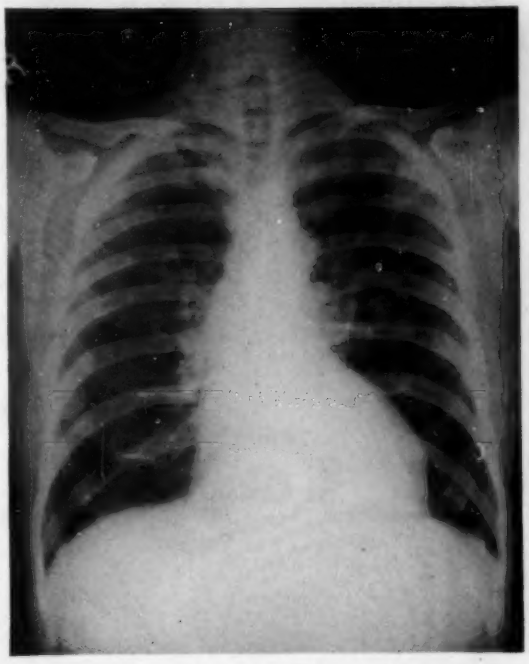


Fig. 3. Hypertension. There are hypertrophy and dilatation of the left ventricle and accentuation of the curve of the ascending aorta. There is increased amplitude of pulsations of the ventricle and aorta.

HYPERTENSION

Increased peripheral resistance calls for more work on the part of the left ventricle. This is met by hypertrophy of the left

Fig. 4. Hypertensive heart disease (glomerulonephritis). Marked left ventricular hypertrophy and dilatation resulting from hypertension. Increase in transverse diameter due to relative insufficiency. Hypertrophy of the right ventricle, shown by elevation of the apex from the diaphragm, indicates that relative mitral insufficiency is of long standing.

ventricular myocardium. The left ventricle becomes thickened and rounded and there is an increased prominence of the curve of the ascending aorta (Fig. 3). Roentgenoscopically, there is increased amplitude of pulsations of the left ventricle and aorta.

As the condition progresses, the heart becomes larger in transverse diameter. This

Fig. 6. Arteriovenous aneurysms at the lung roots associated with patency of the ductus arteriosus. The enlarged vascular trunks exhibit marked expansile pulsations.

indicates a relative mitral insufficiency[10] and, at this time, a systolic murmur will be heard at the apex. The importance of a relative mitral insufficiency is usually not fully appreciated. This change occurs as a result of stretching of the mitral ring, so that the normal flaps no longer suffice to

Fig. 5. Cor pulmonale, in a case of pneumonoconiosis. The pulmonary artery is prominent and there is an increase in the length of the heart. Strong systolic pulsations are observed in the region of the conus and cardiohepatic angle as a result of increased activity of the right ventricle. Note elevation of apex due to right ventricular hypertrophy, and pulmonary changes of pneumonoconiosis.

occlude the enlarged opening. It frequently follows in the course of hypertension, aortic valvular disease, coronary thrombosis, or anemias, and is due to relaxation of the myocardium. While the heart may adjust itself to the change and carry on, more or less effectually for a time, left-sided failure ultimately ensues. It is

Fig. 7. Same case as Figure 6. Some of the enlarged vessels are seen in plane view, others in cross section. Orthoctonous pulsations which were best seen in the oblique positions served to identify the enlarged shadows.

for this reason that we regard the appearance of a relative mitral insufficiency as the first milestone of broken compensation.

In long-standing hypertension, particularly of the so-called "cardiorenal" type, where the relative mitral insufficiency has existed for a long time, secondary changes are noted in the right ventricle. The right

Fig. 8. Primary pulmonary thrombosis. This case shows a loss of convexity of the left border which simulates coronary artery disease. The latter, however, was excluded by roentgenoscopic study. There is an increase in the transverse diameter of the heart which showed pulsations of normal amplitude. Infarct in right lung indicated by arrow.

Fig. 9. Myxedema heart. There is marked symmetrical enlargement. The cardiohepatic angle in this case is acute due to the complicating presence of pericardial effusion. In the absence of fluid, the angle is greater. The amplitude of pulsations is slightly diminished, but in the presence of much fluid pulsations may be imperceptible.

Fig. 10. Same case as Figure 9 two months after thyroid medication. The marked reduction in size is typical of these cases.

ventricular myocardium becomes hypertrophied in consequence of increased pulmonary resistance due to prolonged left-sided failure. This is shown by elevation of the apex from the diaphragm (Fig. 4).

HYPERTENSION OF LESSER CIRCULATION

Under this heading may be grouped several different conditions which operate under similar mechanical influences.

Fig. 12. The heart in a severe case of macrocytic anemia. There is more or less symmetrical enlargement of all chambers. The rate and amplitude are increased.

Cor Pulmonale. Chronic increase in pulmonary tension, such as occurs in pneumonoconiosis, asthma and pulmonary fibrosis, produces profound influences upon the circulation. There is interference with the venous flow into the thorax.[1] The pulmonary conus becomes considerably dilated and the right ventricle hypertrophied. The primary branches of the pulmonary artery

Fig. 11. Thyrotoxicosis. There is a loss of convexity of the left border with an increased prominence of the pulmonary artery. These changes become more prominent during deep inspiration. There is tachycardia, with a marked increase in amplitude of pulsations of the heart and great vessels. The lungs are usually hyperventilated.

are almost always enlarged, and these exhibit expansile pulsations. Increased pulsations are likewise observed in the region of the conus and at the lower right heart border, the latter being due to hypertrophy of the right ventricle. There is usually an increase in the length of the heart and the apex is elevated from the

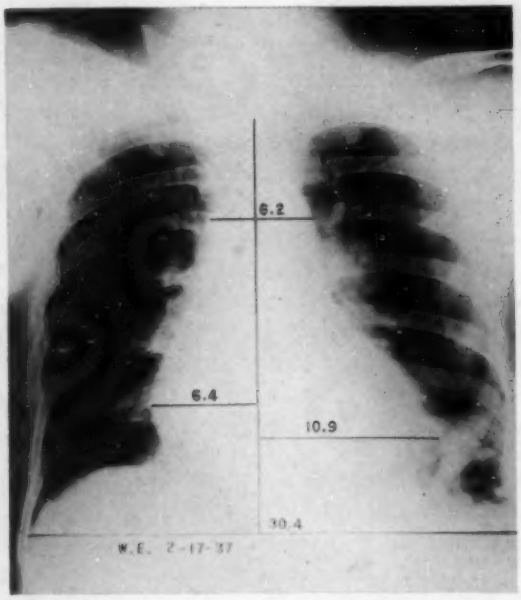


Fig. 13. Avitaminosis (beriberi). All chambers are enlarged. There may be evidence of failure of one or both sides. The rate is increased and pulsations are strong and heaving. (Courtesy of Dr. Soma Weiss, Thorndike Memorial, Boston City Hospital and of *Annals of Internal Medicine*.)

diaphragm (Fig. 5). The associated lung changes are usually obvious and assist in identification of the lesion. As the process continues, the nutrition of the heart becomes progressively impaired as a result of interference with the circulation by the narrowed pulmonary vascular bed. Moreover, alterations in the walls of the alveoli

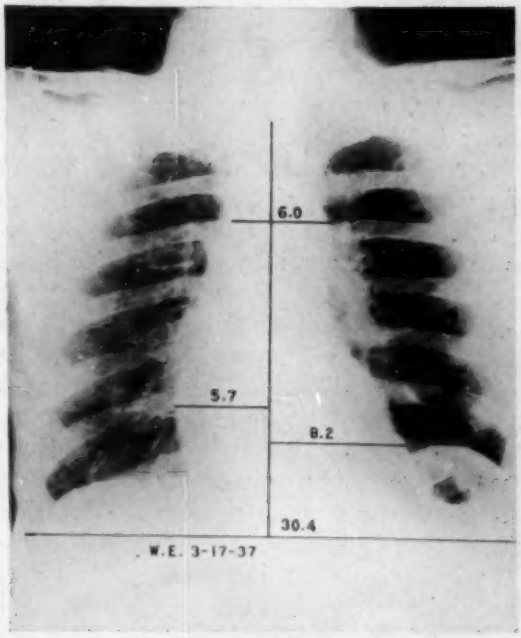


Fig. 14. Same case as Figure 13 one month after administration of vitamin B_1. Heart restored to normal size and contour. (Courtesy of Dr. Soma Weiss, Thorndike Memorial, Boston City Hospital and of *Annals of Internal Medicine*.)

lead to imperfect oxygenation, so that there is a qualitative, as well as quantita-

Fig. 15. Coronary disease. Changes in contour depend upon the acuteness and degree of thrombosis. This is an acute case. Note marked loss of convexity of the left border. The heart appears to sag. There is congestion of the lungs due to left ventricular failure. Roentgenoscopically, the amplitude of pulsations was much diminished (compare with Fig. 18).

tive, change in the blood supply.[9] The forceful beat previously noted gives way to diminished amplitude of cardiac contractions, signifying myocardial impairment and impending failure.

Arteriovenous Aneurysms. The effect of abnormal shunting of arterial blood into the venous system is to diminish the resistance to the output of the left ventricle. At the same time, the work of the right chambers of the heart is increased. This condition not infrequently occurs in the head or extremities. If the aberrant anastomosis is in the pulmonary circuit, a considerable load is thrown upon the right ventricle. Enlargement of the right chambers, however, usually consists of dilatation rather than hypertrophy.[2] Roentgenoscopic examination shows increased activity of the right ventricle and ascending aorta with diminished size and pulsations of the descending aorta. Enlarged anastomotic vessels exhibiting marked expansile pulsations may frequently be seen at the lung roots (Fig. 6). Careful roentgenoscopic examination in the oblique positions is necessary for the proper identification of these shadows which are not infrequently mistaken for enlarged nodes or metastases (Fig. 7).

Pulmonary Thrombosis. This condition may be primary, arising in the pulmonary arteries as a result of localized arteritis produced by infection, or it may be secondary to a lesion anywhere in the venous circuit or right side of the heart. The immediate effect is to increase the burden on the right ventricle. If the degree of right ventricular strain is severe, there will be an increase in the size of the chamber (Fig. 8). In one of our cases which went to necropsy, the electrocardiogram was interpreted as evidence of coronary artery disease. Roentgenoscopic examination, however, excluded coronary artery disease and none was found at autopsy.[4]

Clinically, pulmonary thrombosis is confused chiefly with thrombosis of the coronary arteries and, in these cases, roentgenoscopic examination is of definite value. In pulmonary thrombosis, the increase in transverse diameter of the heart is associated with contractions which are normal or only slightly diminished. Occasionally, a pulmonary infarct may be visualized. In coronary disease, the heart itself shows changes of localized myocardial damage.

Myxedema. Changes in the heart are not invariably found in myxedema, even though thyroid function be greatly depressed. When present, however, the appearance is fairly typical. All four chambers are more or less symmetrically enlarged due to infiltration of the musculature with a mucoid substance.[1] This anatomical change, together with reduced thyroid secretion, probably accounts for the diminished activity of the myocardium. The contour (Fig. 9) resembles the picture of pericardial effusion. In the latter, however, the amplitude of pulsations is much weaker than in myxedema. There will also be noted a difference in the cardiohepatic angle which is more acute in effusion due to bulging above the point of attachment of the pericardium. Not infrequently, however, pericardial effusion accompanies myxedema and, in these cases, differential diagnosis from the appearance of the heart alone may be difficult. Nevertheless, the striking response of myxedema to thyroid medication is characteristic of the disease, the heart being reduced to normal size and contour in a comparatively short time (Fig. 10).

Thyrotoxicosis. We have frequently been able to suspect the presence of hyperthyroidism by the appearance of the heart, the most important evidence being obtained by roentgenoscopy. The rate is rapid and the amplitude of contractions of all components of the cardiovascular shadow is increased. There is a loss of convexity of the left heart border probably due to a relative anoxemia of the myocardium. This is associated with a prominent pulmonary conus and hyperventilation of the lungs (Fig. 11). In these cases, we invariably examine the neck for evidence of tracheal displacement or compression due to an

enlarged cervical or substernal thyroid. Thyrotoxicosis is one of the common causes of auricular fibrillation[7] so that particular note should be made of the rhythm in suspected thyrocardiacs.

Anemias. In the chronic anemias there is enlargement of all the cardiac chambers (Fig. 12) and the amplitude of pulsations is increased. The latter observation is of importance in differentiating symmetrical enlargement due to other causes, as, for example, myxedema and hypertensive failure, in which the force of pulsations is usually either normal or diminished. It has been our observation that conditions associated with a general anoxemia of the heart muscle, except in the terminal stages, usually show an increased rate and amplitude of cardiac contractions. This does not hold true in coronary artery disease, for, in this case, there is a localized ischemia.

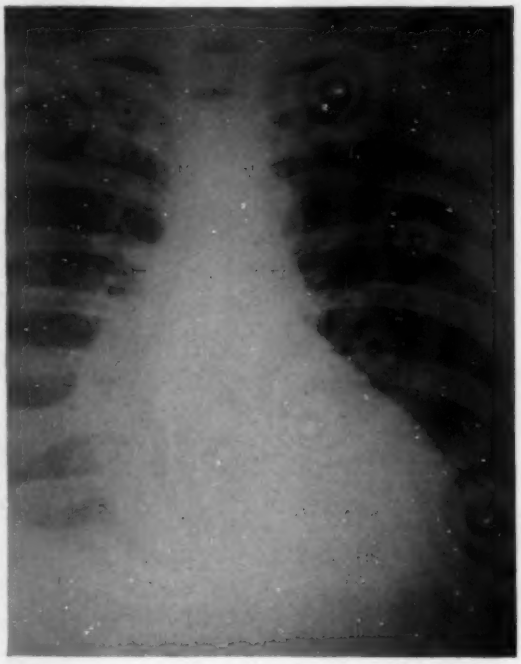


Fig. 16. Chronic hypertension with coronary thrombosis and infarction. Note left ventricular hypertrophy due to hypertensive state. This prevents sagging of left border observed in absence of hypertension. Instead, there is a localized area of flattening corresponding to site of infarct (brackets). Pulsations were markedly diminished in this area (compare with Fig. 17).

Fig. 17. Same case as Figure 16. Kymogram showing localized diminution of contractions in area of infarction (brackets) which was observed roentgenoscopically.

Avitaminosis. The changes in the heart produced in beriberi are a striking manifestation of vitamin deficiency. Weiss and Wilkins[11] have shown that the "wet" form of this disease is associated with hydropic degeneration of the muscle fibers. The heart is more or less symmetrically enlarged (Fig. 13) so that it appears to be a large heart of normal contour. Severe forms of this condition are invariably associated with failure of either or both ventricles, so that congestive infiltration of the lungs, or signs of tricuspid insufficiency, or both, will be observed. Roentgenoscopically, the heart shows an acceleration of rate, with a marked, generalized increase in amplitude. These cases show a striking response to vitamin therapy, the parenteral or oral administration of vitamin B_1 restoring the heart to normal size and contour in a comparatively short time (Fig. 14).

Coronary Artery Disease. Occlusion of a coronary artery may be acute or gradual, the latter form of thrombosis being more

Fig. 18. Coronary disease. Long history of repeated mild attacks. The contour of the heart is normal in appearance. Roentgenoscopic examination showed an area of localized myocardial damage due to infarction (compare with Fig. 15).

common. As the lumen of the vessel is shut off, there is an area of localized ischemia in the heart muscle. This results in an immediate loss of tone. Within twenty-four hours, there is necrosis of the myocardial fibers.[8] In consequence, the normal convexity of the heart borders is diminished (Fig. 15), and the activity of the involved portion of myocardium greatly reduced.[6] These changes appear early, and are therefore of distinct value in recognizing the earlier stages of the disease. Occasionally, a calcified coronary artery may be demonstrated.[13] This, however, is a late manifestation of coronary involution and, it must be remembered, coronary sclerosis may occur without occlusion or the formation of an infarct in the heart muscle. It is therefore necessary to carefully scrutinize the component parts of the cardiovascular silhouette for evidence of localized diminution in the force of contractions. This indicates local myocardial damage and may be regarded as good inferential evidence of thrombosis.

Hypertension frequently precedes coronary artery disease[7] and since it produces alterations in the contour and activity of the heart, the presence of both lesions gives rise to a slightly different picture.[3] It is recalled that the amplitude of contractions in hypertension is increased. With the advent of thrombosis, the force of contraction is diminished, so that it may equal, in intensity, the force of contraction of a normal heart. But, if we can establish the presence of pre-existing hypertension by virtue of the left ventricular hypertrophy, then we must regard the force of beat as being diminished; in other words, it is too weak for this particular heart and indicates myocardial damage (Fig. 16). If an infarct has formed, careful examination will show a localized area of flattening of the ventricular contour in the region where the amplitude of contractions is diminished (Fig. 17).

If the process of occlusion is very

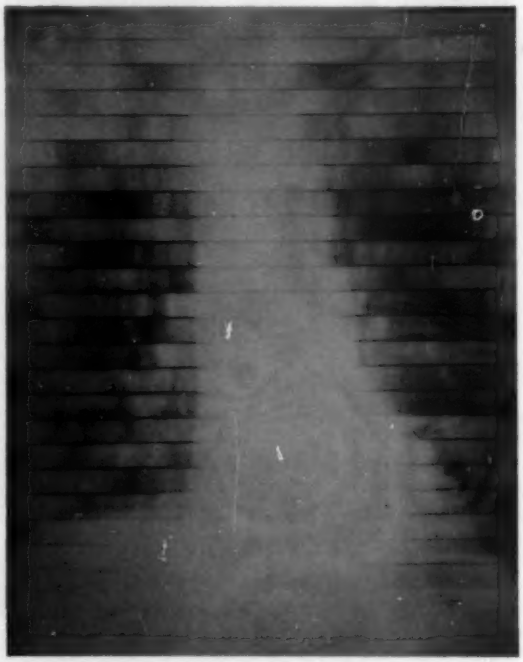


Fig. 19. Same case as Figure 18. Kymogram showing localized diminution in amplitude of ventricular contractions.

gradual, so that sufficient time is allowed for the formation of collateral circulation, there may be no alteration in the contour of the heart, which will therefore present a normal appearance on the roentgenogram (Fig. 18). Roentgenoscopic examination, however, will usually detect a localized area of diminished amplitude indicating local myocardial damage (Fig. 19). Roentgenoscopic study is a valuable adjunct in the diagnosis of coronary artery disease, for, by this means, we have frequently been able to establish a diagnosis before positive electrocardiographic findings were in evidence.

SUMMARY AND CONCLUSIONS

Non-valvular disease of the heart and various forms of myocardial insufficiency frequently produce distinctive roentgenological appearances. Careful roentgenologic study is therefore well suited to the identification and appraisal of disturbed cardiac dynamics. Its importance is further increased by its ability to depict the relationship of the greater and lesser circulation in the individual case. It offers confirmatory evidence of a diagnosis established by other clinical methods and frequently assists in the differentiation of the causes of cardiac enlargement and decompensation.

REFERENCES

1. Boyd, W. Pathology of Internal Diseases. Lea & Febiger, Philadelphia, 1935.
2. Holman, E. Arteriovenous Aneurysm. Macmillan Co., New York, 1937.
3. Levene, G. Further observations on the roentgen diagnosis of coronary disease. *Radiology*, 1938, *30*, 309–319.
4. Levene, G., Lowman, R. M., and Wissing, E. G. Roentgenologic and electrocardiographic diagnosis of coronary disease; comparative study of 140 cases. *Am. Heart J.*, 1938, *16*, 133–142.
5. Levene, G., and Reid, W. D. Differential diagnosis of organic heart disease by the roentgen ray. Am. J. Roentgenol. & Rad. Therapy, 1932, *28*, 466–480.
6. Levene, G., Wheatley, F. E., and Matthews, H. Roentgen diagnosis of coronary disease. Am. J. Roentgenol. & Rad. Therapy, 1934, *31*, 588–592.
7. Levine, S. A. Clinical Heart Disease. F. A. Davis Co., Philadelphia, 1934.
8. Mallory, G. K., and White, P. D. Further observation on the healing of myocardial infarction. *New England J. Med.*, 1937, *217*, 1110–1112.
9. Oppenheimer, B. S., and Hitzig, W. M. Use of circulatory measurements in evaluating pulmonary and cardiac factors in chronic lung disorders. *Am. Heart J.*, 1936, *12*, 257–271.
10. Reid, W. D., and Levene, G. Roentgenologic consideration of mitral heart disease. *New England J. Med.*, 1932, *206*, 1027–1032.
11. Weiss, S., and Wilkins, R. W. Nature of cardiovascular disturbances in nutritional deficiency states (beriberi). *Ann. Int. Med.*, 1937, *11*, 104–148.
12. White, P. D. Weakness and failure of left ventricle without failure of right ventricle. *J. Am. M. Ass.*, 1933, *100*, 1993–1998.
13. Wosika, P. H., and Sosman, M. C. Roentgen demonstration of calcified coronary arteries in living subjects. *J. Am. M. Ass.*, 1934, *102*, 591–593.

DISCUSSION

Dr. George W. Holmes, Boston. Dr. Levene has talked at considerable length on the physiology and anatomy of the heart as well as given a discussion of the roentgenologic findings. I am going to leave the first part to Dr. Levine.

Based on his knowledge and experience, Dr. Levene has described the roentgen signs which should be present. Roentgenologists, however, I am convinced, will find these data difficult to obtain and many times completely absent. I would not personally make a negative diagnosis of cardiac disease from the roentgen findings, and it is my belief that the diagnosis is oftentimes impossible by any and all methods. It is therefore important that the roentgenologist be cautious in drawing conclusions from the data which he obtains. The observations should be carefully made, the data obtained recorded. The amount and value of these data will be considerably increased if, as Dr. Levene has observed, the observer has a complete knowledge of cardiac disease. If one knows what to look for, one is more likely to see it. On the other hand, conclusions from these data, which are necessarily incomplete, should be most conservative.

Dr. Samuel Levine, Boston. I have learned during the past few years that the roentgen ray had something more to offer than I origi-

nally suspected in the diagnosis of coronary artery disease, but I doubt very much whether we can rule out coronary artery disease by the roentgen ray.

Angina pectoris—I mean by that the lethal form that was described by Heberden, the kind that can kill instantly—can occur from one small plaque in a coronary artery without any other changes in the heart muscle. The heart can be normal in size, normal when you weigh the heart at autopsy, with no dilatation; no alteration in blood pressure; rhythm normal; no murmurs; in fact, the most innocent sounding of all hearts, the kind of heart that baffles the life insurance companies. They will pass that sort of patient for a hundred thousand dollars of life insurance and the next week he may drop dead of angina pectoris. Yet there need be no more pathology than a small plaque, 2 cm. from the mouth of the left descending coronary artery.

I feel that it would be inconceivable in some of them to expect alterations in the integrity of the muscle, that could be seen by the roentgen ray. There are many that have myocardial changes—myocardiac disability, fibrotic developments, thinning of the walls—and naturally, in the course of time, changes that have just now been shown. But I doubt very much whether they all do that. Angina pectoris is a useful term. It tells a great deal. Yet the diagnosis is made entirely on the history. All the patient needs to have is a certain type of distress, a sternal constricting feeling brought on in the proper fashion upon effort, with radiation or without it. That is angina pectoris and that is angina pectoris about as certainly as tuberculosis is diagnosed when you find the tubercle bacillus.

The diagnosis of angina pectoris is accurate; it is altogether too accurate. I wish it were not as accurate as it is. When I look over the follow-up notes on the cases I see diagnosed as angina pectoris, they all progress according to an expected pattern. You occasionally need to add a question mark after "angina." But when the decision is definitely made, there is no doubt about it. Yet it requires very little pathology to produce it. Therefore, I would caution ruling out the diagnosis of coronary artery pathology or angina pectoris on the basis of roentgen examination. Rule it in on the findings that you have heard, but I do not see how one could at the present time rule it out.

I want to draw a distinction between angina pectoris and coronary artery disease. You can have coronary artery disease without angina pectoris. We have seen cases that were visualized as having coronary sclerosis, but there was no angina pectoris. The two are different. In such cases the muscle is not anoxemic. The vessels were calcified but the patient was getting all the blood that he needed. The two are very closely related but not synonymous. You may have coronary arteriosclerosis without angina pectoris. In fact, you can have angina pectoris in rare circumstances without coronary arteriosclerosis.

In aortic stenosis alone, without any disease of the coronary arteries, those with marked buttonhole aortic valves, you will find angina pectoris commonly. When it occurs in aortic stenosis it is just as real as the ordinary angina of coronary artery origin. These patients die instantly in the same fashion and they have the same kind of pain. The aortic cases of the older group often have some coronary artery pathology, too, but we have some younger ones in the twenties who had normal coronary arteries, but had angina pectoris and had a sudden death that characterized the disease.

Therefore, I think the term "angina pectoris" is very valuable and makes a worthwhile clinical diagnosis, but it denotes a physiological potentiality, a state of the heart. We do not yet know the exact reason for the attacks in angina pectoris, and why it occurs at 9:15 and not at 9:30 or 9:00 o'clock. There is a good deal about angina pectoris that we do not know, but we can recognize it clinically and give it a name. It is generally associated with coronary artery disease. Rarely is there another mechanism behind it.

I wonder how often the flat left ventricle is seen without angina pectoris or without coronary artery disease. It would be very helpful to know the limitations of that sign. I have seen Dr. Levene work and I know that he has seen things that were important in the roentgenogram that were not being talked about or discussed previously, and I have a good deal more regard for the roentgen-ray clues that you get in coronary artery disease than I previously had, but I would like to emphasize an element of caution.

Then there is another point which I would like to make, which I hope will not be taken amiss. I think we should be cautious about

physiological explanations of things unless we are full-fledged physiologists. In hypertension, the visible contraction of the left ventricle is not greater than in the normal heart. The movement of the left ventricle in hypertension is often less than in the normal heart. The fact that the pressure is 240 mm. does not mean that the heart has to go through a greater excursion to contract. Many of the patients with hypertension have diminished apex impulses clinically, and I wonder whether that is not so fluoroscopically. The apex impulse is often diminished in hypertension. You can raise the pressure to 240 from 125 diastolic with a small movement. The largest amplitude we see in hyperthyroidism, where there is a normal pressure but a dynamic heart. The heart snaps with a lot of leeway between diastole and systole.

Then, terms like an increase in the work of the heart from increasing the peripheral pressure in arteriovenous aneurysm, there is something wrong with this. The peripheral pressure in arteriovenous aneurysm is not increased though the work of the heart is increased. The peripheral pressure in arteriovenous aneurysm, if anything is diminished. The heart dilates because the same blood is coming through more frequently. There is a shunt; work depends upon blood volume as well as blood pressure. The left ventricle is not ejecting blood at an increased pressure although the total work of the heart is increased.

I think the roentgenologist will be stepping on dangerous ground if he goes far afield into physiology unless he masters the subject, too. I would like to make that comment because I think such work would be more acceptable if it is not vulnerable in the physiological sense.

ROENTGENOLOGY OF CONGENITAL CARDIOVASCULAR DISEASE*

By HUGO ROESLER, M.D.
PHILADELPHIA, PENNSYLVANIA

THE roentgenologist should hold the position of a consulting physician. Hence it may be of interest to present in the beginning some facts pertaining to the clinical aspects of congenital cardiovascular disease. The incidence is perhaps 1.5 per cent out of all cases with organic heart disease and definitely higher if children and adolescents only are under consideration. When signs of heart disease are found before the age of twenty, the diagnosis usually lies between a rheumatic and a congenital lesion. It is a mistaken idea that congenital heart disease is observed only or mainly in the young and that cyanosis and the presence of murmurs are leading diagnostic features. Clinical and roentgenological signs are often inconclusive in infants and young children. After puberty these signs are more characteristic and the identification of the actual lesion becomes therefore easier. Such a diagnosis aids considerably in the prognostication of the case. Generally speaking, there is a high mortality before the fourth decade. The average expectation of life in the group without cyanosis is the period between puberty and the middle thirties. A good number of cases reach a ripe age. Of those more common anomalies which usually can be identified, conditions such as patency of the ductus arteriosus, interventricular and interatrial septal defects, right-sided aortic arch and coarctation of the aorta are compatible with a perfectly normal life. The average expectation of life in the group with cyanosis is the period between childhood and adolescence, with only a very few reaching a mature age. The most important complication is subacute bacterial endocarditis and endarteritis; it is responsible for the death of 1 out of 6 cases. Pulmonary tuberculosis is not particularly common.

Prior to the advent of cardioroentgenology a good many cases were not diagnosed or passed through life classified as heart disease of rheumatic etiology. In our material we found positive roentgenological findings in 85 per cent of all cases; they were of definite diagnostic value in 51 per cent. Abnormalities in the pulmonic area were approximately three times more frequent than in the aortic area.

For didactic purposes we may speak of four groups of roentgenological aspects of structural anomalies.

First, they may exist without any roentgenological indication of their presence. An example is the isolated interventricular septal defect of rather small size where the clinician finds a harsh systolic precordial murmur, often accompanied by a thrill.

Second, there are deviations from the normal which are not characteristic for a congenital structural anomaly. We refer, for instance, to a general cardiac enlargement which is noted in the presence of a two- or three-chambered heart, or with the origin of a coronary artery from the pulmonary artery; another example is presented by an abnormal prominence of the pulmonic arch, as noted, with patency of the ductus arteriosus or with pulmonic regurgitation. It is obvious that general cardiac enlargement or prominence of the pulmonic arch occurs in a number of acquired diseases.

In a third group we would place some conditions which reveal fairly characteristic features such as: (1) The *coeur en sabot* or wooden shoe heart: the left lower contour is blunt (it has been likened to a sheep's nose) and formed in part by the right ven-

* From the Department of Radiology, Temple University Medical School and Hospital, Philadelphia. Read in a Symposium on Heart Disease at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938.

tricle. In the lateral view the mass of the heart is noted to protrude ventrad. Electrocardiographically right axis deviation is present and clinically cyanosis and clubbing are observed, as a rule. Anatomically one finds pulmonary artery or conus stenosis, associated with a marked degree of right ventricular hypertrophy, an interventricular septal defect and a dextroposition of the aortic orifice. (2) The large globular heart with marked enlargement of the pulmonary conus, artery and its branches, and with a small aorta. Roentgenoscopically the pulmonary artery and its branches usually reveal exaggerated pulsations. The roentgenographic aspect of the enormously enlarged hilar vessels has been mistaken as evidence of lymph node enlargement, tuberculous or malignant in nature. Electrocardiographically a lesser degree of right axis deviation and, not uncommonly, auricular fibrillation are noted. Clinically one observes physical underdevelopment, and the diagnosis of mitral valvular disease is often made; actually, the combination of these two lesions—one congenital and the other one acquired—is not rare. Anatomically one finds an interatrial septal defect, associated with marked dilatation of the right heart cavities, of the pulmonary artery and its branches and hypoplasia of the aorta. (3) The prominence of the cardiac silhouette to the left in the anterior view, and dorsad in the left anterior oblique view, both findings indicating left ventricular preponderance. Electrocardiographically there is present left axis deviation, and clinically one notes cyanosis and clubbing. Death occurs early in life. The anatomical finding consists in atresia or hypoplasia of the tricuspid orifice together with a defective development of the right ventricle.

Finally, in a fourth group belong structural anomalies with unequivocal roentgenologic findings. We cite the following examples: (1) The cardiac shadow is mainly to the right, with no mechanical factors accounting for it. The right leaf of the diaphragm is lower in position than the left one. The other organs of the body reveal a normal situs or occasionally a partial transposition. The aortic shadow indicates a normal left-sided or abnormal right-sided course. Electrocardiographically different patterns are observed; the clinical findings may vary between normal functional capacity and the picture of cardiac invalidity with cyanosis. Anatomically one never observes the architecture to show a complete, mirror-like inversion, but partial inversions associated with other congenital cardiac defects are found. (2) Small notches or serrations along the lower margins of the dorsal portions of the ribs are observed, and the aortic arch, as visualized in the left anterior oblique view, reveals an indentation or discontinuity. Electrocardiographically findings compatible with systemic hypertension are noted; clinically one observes first, hypertension limited to the upper part of the body, and second, an arterial collateral circulation about the trunk. The anatomical finding consists in a localized stenosis in the distal portion of the arch, also called coarctation of the aorta. The enlarged and tortuous intercostal arteries, as a part of the arterial collateral circulation, are responsible for the defects along the ribs. (3) The aortic knob is atypically noted in the region of the right sternoclavicular junction and at this level esophagus and trachea deviate to the left instead of, as normally, to the right. In the right anterior oblique view esophagus and trachea deviate ventrad, as a rule, with a round shadow mass noted dorsad. The presence of an abnormal shadow mass in the upper mediastinum, together with the deviation of the trachea and esophagus has repeatedly led to the erroneous diagnosis of aneurysm arising from the aortic arch or innominate artery, mediastinal glandular mass, or substernal goiter. Electrocardiographically nothing particular is observed and clinically the condition may escape recognition altogether; very occasionally, however, dyspnea, dysphagia and laryngeal palsy may result from pressure. The anomaly referred to is the per-

sistent right-sided aortic arch which crosses over the right bronchus and, usually recrossing to the left, displaces esophagus and trachea. Where arch and descending aorta unite a diverticular enlargement is usually found. The heart often reveals no other malformation.

DISCUSSION

DR. GEORGE W. HOLMES, Boston. As we would expect, Dr. Roesler has presented a sound and complete discussion of his subject. It would be absurd for me to attempt to add anything to it. I would like to emphasize, however, some things that Dr. Roesler knows and many of us do not, and one of them is that before anyone attempts the diagnosis of these conditions, he must have not only a sound knowledge of anatomy and pathology of the heart, but he must also know embryology and development. I do not know about the other medical schools but in the community in which I reside, our students have a very meager knowledge of embryology. It may be fairly good in the other branches. Before we attempt to equal or perhaps I should say emulate Dr. Roesler, we should spend a considerable time in the study of embryology and development of the heart and its great vessels.

DR. SAMUEL LEVINE, Boston. I would like to make a few clinical comments on congenital heart disease. For some curious reason, there is an undue amount of rheumatic heart disease in patients who have congenital heart disease. The combination of both seems to be more common than could be accounted for accidentally. I do not know why that is so. We have, therefore, the double problem at times to diagnose the congenital heart defect plus the acquired rheumatic lesion.

Congenital cardiac disease is not infrequently associated with mental retardation and also with lack of physical development. That is easily comprehensible. In some of the congenital defects, there is a constant diminution in the amount of oxygen going to the entire body, especially to the brain. If there is, for instance, a significant shift of arterial blood going through a patent ductus into the pulmonary system, that child can grow up (and I have seen 2 such cases) with a constantly smaller supply of oxygen going out into the periphery. Those cases need not at that time compensate for it by the polycythemia that often develops later on. Therefore, if there were anything that should ever become available in the way of treatment, those are some of the considerations that we would have to keep in mind—the prevention of lack of development, the prevention of subacute bacterial endocarditis or any form of bacterial endocarditis which accounts for the fatalities in 25 per cent of the cases of patent ductus.

The polycythemia in congenital cardiac disease can be very marked, with a red blood cell count of ten million. We had a boy whom we diagnosed as having a trilocular heart. We failed to obtain an autopsy to prove it, but that was the most likely diagnosis. He had no ventricular septum, or a huge defect in the ventricular septum, who was a very handy donor at the Brigham Hospital for many years before liver was discovered as a cure for pernicious anemia. He used to transfuse our anemia cases. He would come in about once every two or three months and bleeding always gave him subjective relief. He would have about twelve or thirteen million red cells and one phlebotomy would do for two patients.

It is well to keep in mind that cyanosis of congenital heart disease is a very simple matter. It is not of the same origin as cyanosis in acquired heart disease. Cyanosis of congenital heart disease is due to a right to left shunt; venous blood goes to the left heart without going to the lung. It is not because of any lack of aeration of the lungs. When it is of a certain amount, it will result in gross cyanosis and when it is prolonged enough and marked enough, there will be a polycythemia and clubbed fingers.

Vol. 42, No. 1

ROENTGEN EXAMINATION OF THE AORTA AND PULMONARY ARTERY*

By MARCY L. SUSSMAN, M.D.
The Mount Sinai Hospital, Service of Dr. Leopold Jaches
NEW YORK CITY

IT IS proposed at this time to review the roentgen methods of investigation of the aorta and pulmonary artery, and particularly to discuss their value in early abnormalities. The report will limit itself to the objective findings of the roentgen examination and will exclude what may be inferred by correlation with the clinical history.

The roentgen study of the aorta should include investigation of its size, with special note of any localized enlargement, of its shape, course, roentgen density, pulsation and mobility. Modern practice requires that the investigation should be made in at least posteroanterior, right and left oblique and lateral positions. Mensuration is important but must be practiced only under conditions where the projection error is minimal, that is, either from orthodiagraphic tracings or from teleroentgenological studies. A standard position of the diaphragm and exclusion of chest deformity is just as important in this type of mensuration as in the usual cardiac measurement, although the literature does not emphasize it. Roentgenography must be supplemented by roentgenoscopy for the study of pulsation, mobility, density and course. Teleroentgenoscopy, as I believe Dr. G. W. Holmes still practices it, appears to be the ideal method, but ordinary roentgenoscopy in experienced hands is quite satisfactory. In general, during roentgenoscopy the patient should be studied through all stages of rotation and that position chosen for roentgenography which yields the information desired.

I have emphasized the need for experience in roentgenoscopy because the size and shape of the chest and the position of the diaphragm play such an important rôle in determining the apparent size and shape of the aorta. Furthermore, the roentgenoscopic image, particularly in the oblique positions, is magnified by a short tube-screen distance and a relatively large object-screen distance, so that the likelihood of error is great and may be minimized only by the experience acquired in the examination of a large number of cases, normal and abnormal.

AORTA

Size. The most definite evidence of aortic disease obtained roentgenologically is that of increase in size, local or diffuse. Except in unusual circumstances, such as where well-defined atheroma results in a marked increase in density, a direct measurement of the aorta cannot be made. Since measurements are indirect, several methods are available. They are subject to error because of individual anatomical and pathological variations. As a first approximation of the general over-all dimension, the transverse diameter of the superior mediastinal shadow in the posteroanterior view may be taken. This is best determined as a summation of the widest supracardiac shadow to the right and the widest portion of the aortic knob to the left. When this measurement exceeds 5.0 cm. further investigation is desirable. An estimation of the length of the aorta between its origin and its arch can be made by observing the space between the aortic knob and the inferior margin of the clavicle, which ordinarily should not be less than 2.0 cm., as well as by the distance between the superior margin of the arch and the level of the junction of the right cardiac and supracardiac contours.

The diameter of the ascending aorta may

* Read in a Symposium on Heart Disease at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938.

be estimated by turning the patient into the right oblique position under roentgenoscopic vision until the shadows of ascending and descending aorta are superimposed. The normal values given are 2–3 cm. Theoretically, this measurement should be possible at all times; practically, the inner margin of the ascending aorta is not well defined and the measurement must be taken from the edge of the left main bronchus. Error may also result because the entire area may be obscured by the superior vena cava. In the posteroanterior view, the outline of the right supracardiac shadow should not bulge farther to the right then the right cardiac shadow. When it does, a dilatation of the ascending aorta can be assumed. Measurements of the ascending aorta have also been made from the left oblique position, the patient being rotated until a clear aortic window is seen. Reich[1] measures the distance from the right main bronchus to the contour of the ascending aorta just above the cardiac shadow. Average figures are 4 cm. Hampton and Jones measure a similar diameter, the inner limit being within the aortic window. Measurements under 5.5 cm. are given as normal for this diameter. The use of the pulmonary artery to define the border of the aorta in the left oblique position has not been particularly valuable in practice because of the difficulty in visualizing the artery itself.

The diameter of the arch may be measured either directly when its density is sufficient, or indirectly, from the trachea or barium-filled esophagus to the contour of the knob. When the aorta is elongated these mediastinal structures do not maintain their usual relationship with the arch and it may be necessary to turn the patient into a more or less right oblique position to obtain a closer approximation. This diameter measures 2–4 cm. with an average of 2.5 cm. for the younger age groups; 2 mm. is allowed for the thickness of the wall of the trachea or esophagus. The descending aorta can be visualized ordinarily in either oblique position or in the lateral view and when dense enough permits of direct measurement.

Hampton[4] states, with considerable evidence, that the average normal diameter of the arch as seen roentgenographically is 3.5 cm. and that of the ascending aorta is 4.5 cm., the difference from the normal postmortem figures of 1.8–2.0 cm. being due to dilatation by the circulating blood. Vaquez and Bordet have stated that a measurement 5 mm. in excess of what is average at a patient's age is definitely abnormal and a measurement 1 cm. above normal indicates considerable enlargement. When the measurements are made by the same individual under the similar conditions these figures are probably acceptable.

Pulsation and Mobility. Pulsation must of necessity be studied by roentgenoscopy or kymography. Roentgenoscopically, increase in pulsations occurs in hypertension and in aortic insufficiency, whereas arteriosclerosis and aneurysm are ordinarily associated with diminution. However, kymography has shown quite clearly that the movement seen roentgenoscopically is the resultant of the increase in width of the tube during pulsation and displacement in position of the tube as a whole. In the ascending aorta there is a further complication since systolic contraction of the left ventricle draws the ascending aorta along with it so that the recorded pulsation is the resultant movement of intrinsic distention and of displacement. It is not surprising, then, that in a large number of normal individuals, in the posteroanterior view, the roentgen kymogram reveals a rapid medial movement in the region of the ascending aorta during systole when we should expect a lateral expansile movement. In other cases, the kymogram shows practically no pulsations in this position because the two movements of distention and displacement neutralize one another. The roentgen kymogram has therefore given the reason why study of the aortic pulsations roentgenoscopically does not yield more useful diagnostic information.

Mobility also plays an important rôle in

determining the final form of the kymographic wave. The presence of peri-aortic inflammation and adhesions would naturally tend to modify the displacement factor in resultant curves.

It must be concluded that roentgen kymography does not offer particularly valuable information in the early diagnosis of aortic disease and that the information gained from the study of pulsations roentgenoscopically may be misleading.

Density. The density of the aorta is dependent upon the cross-section of the tube in the direction of the central ray as well as on the structure of its wall. The larger the tube and the thicker and more calcified its wall, the denser it appears. Since in the frontal view, ascending and descending aortas superimpose, density is best studied in the oblique and lateral views. In general, density does not permit of precise measurement.

The roentgen examination of the aorta can therefore give fairly accurate information regarding its size, particularly of its arch. Pulsatility and density are less accurately determined. None of the information is precise, and it will therefore be readily appreciated that in the case of slight variation from normal the possibility of error is considerable. Furthermore, a combination of diseases is not uncommon and may further complicate diagnosis.

Atherosclerotic Aorta. During the late middle and older age periods the aorta becomes atheromatous and elongates. This is manifested anatomically by actual lengthening and consequent tortuosity between two relatively fixed points, the base of the aorta and the aortic hiatus of the diaphragm. The aortic arch reaches higher in the chest so that the space between it and the clavicle is diminished or disappears. The aortic knob appears farther to the left. There may be a bulge of the contour of the ascending aorta to the right in the frontal view, although ordinarily not beyond the outline of the right cardiac contour. The shadow of the descending aorta which ordinarily passes behind the pulmonary artery or conus, bulges more or less to the left of these structures. In the right oblique view, the ascending and descending limbs of the aorta are separated with but slight rotation of the patient, and tortuosity of the descending aorta may be visualized in this position. In the left oblique position the arch of the aorta rises high under the sternoclavicular joint and bends posteriorly. The aortic window is wide and the aorta overlaps the spine. In the frontal position the transverse diameter may be considerably enlarged and entirely out of proportion to the diameter of the aorta as determined by the other methods described above. However, it is not uncommon to find moderate dilatation present in addition to the elongation. Calcification, particularly in the arch, can usually be visualized on a roentgenogram made with a rapid exposure and may be confirmed by roentgen kymography.

Pulsations appear diminished because: (1) periaortic fibrosis fixes the vessel to surrounding tissues; (2) sclerosis and atheroma of the wall reduce its flexibility; (3) if the differential blood pressure within the aortic tube is lessened, the displacement of the tube as a whole is reduced; (4) dilatation to any degree tends to reduce pulsation because the effect of systole is diminished. Pulsation of the left ventricle, however, remains relatively normal. Where pulsations of the aorta are increased, with elongation, tortuosity and increased density, the changes suggest that there is little periaortic fibrosis.

Hypertension. In hypertension the aorta is of normal or perhaps moderately increased diameter as determined roentgenographically. Pulsations ordinarily appear increased. The dilatation is sometimes not confirmed by post-mortem examination and the presumption, therefore, is that the dilatation is functional. We have observed a similar dynamic dilatation in two cases of rheumatic endocarditis in which at post-mortem there were no pathological changes in the aorta. In aortic insufficiency, pulsations are more marked and dilatation is moderate.

Luetic Aortitis. Characteristically, lues affects the base of the aorta and then extends to involve the aortic valve or produces an aneurysm in the ascending portion or arch. The diagnosis of luetic aortitis primarily is based upon the demonstration of dilatation and the ease of diagnosis is proportional to the degree of dilatation.

It has been said that early luetic aortitis is associated with localized increased pulsations of the ascending aorta. More likely, in a diseased, thickened portion of the aorta, diminution in pulsability is to be expected because of reduced distensibility, dilatation and more or less fixation. An apparent increase in pulsations might be due to an increased ventricular factor in the absence of periaortic adhesions. Bordet and Fischgold have described a localized irregularity in pulsation found kymographically in aortitis. We have not been able to confirm these observations but our series is small.

While there is no question that roentgenography often provides the only means of diagnosis of luetic aortitis, it must be emphasized that its early diagnosis cannot be made roentgenologically in most cases. It would be expected that the coronary ostia will be narrowed in many cases before the evidence of aortitis is clear. Kymographic studies[8] on cases of luetic aortitis confirmed this impression in some instances.

Aneurysm. Aortic aneurysm may be diffuse or sacculated. The diagnosis is made by the demonstration of a dilated segment of the aorta or the demonstration of a mass which is part of the aorta. Careful roentgenoscopy is the only satisfactory method of diagnosis. Regardless of the patient's position, the mass cannot be separated from the aorta nor can the shadow of the aorta be seen through it. An aneurysm of the ascending aorta produces a bulge of the right supracardiac shadow laterally and retrosternally. The shadow of an aneurysm of the arch extends to the left, right and upwards. An aneurysm of the innominate artery increases the width of the superior mediastinum to the right. Trachea and esophagus are displaced. A similar process in the descendens produces a mass bulging into the left lung field. Increased density also appears because of increased size of the tube and thickening of the wall. Occasionally the wall is calcified. Some authors have noted a haziness in outline presumably due to these same cases. Multiple aneurysms may occur and produce a lobulated shadow. Dissecting aneurysm is rare. Its roentgenographic appearance is not characteristic except that Canigiani comments on the uniform density due to the collection of blood about the vessel. Theoretically, when the aneurysm is not part of the ascending aorta it should be possible to demonstrate luetic aortitis in this segment of the aorta, but this cannot always be done. When the aneurysm is of arteriosclerotic origin the other portions of the aorta show the tortuosity and dilatation expected in this condition. In the absence of aortic insufficiency, the heart in lues is usually not enlarged.

As a general proposition, pulsations of aneurysm are diminished or absent. When marked aneurysmal pulsation is present there is usually another explanation, such as transmitted pulsation from the heart. A large intrinsic pulsation is not to be expected, since, most important in aneurysm, are the reduced distensibility of the wall and dilatation of the tube. Both factors produce restricted motion. The movement seen roentgenoscopically is more a displacement in position than an increase in width. For this reason absence of pulsation should not necessarily be attributed to the presence of thrombosis. The aneurysm follows the motion of the segment of the aorta in which it lies and the extent of its motion depends on that of the surrounding structures. This is true also for aneurysm-like tumors, so that differentiation on the basis of expansile as contrasted with transmitted pulsations is not often a practical possibility.

THE PULMONARY ARTERY AND ITS BRANCHES

In the posteroanterior view the mid left cardiac contour is formed by the main stem of the pulmonary artery. This passes upwards and backwards merging into the aortic shadow, in the arch of which it divides into right and left main branches. The right passes outwards behind the ascending aorta merging into view at the right hilum. The left pulmonary artery passes backwards and to the left and comes into view at the left hilum. Usually an increase in the size of the pulmonary artery is manifested by an increase in the length, convexity and lateral bulge of the mid left cardiac contour together with an increase in density. In the right oblique position, the pulmonic segment bulges into the retrosternal space: the right main branch may be seen coursing horizontally as a knob from left to right across the posterior mediastinum. In the left oblique position the bulge is into the aortic window: a cross-section of the pulmonic artery may be seen below the arch of the aorta and the left main branch descends broadly into the left lung field. Pressure exerted upon the esophagus either directly or indirectly through the left bronchus is evidenced by an exaggerated indentation shown below that of the aorta.

Dilatation of the pulmonary artery branches is estimated by direct comparison of the central and peripheral vascular pattern with the normal. By rotation in both oblique positions the large hilum branches are better visualized. The left and right main branches are approximately 10 to 13 mm. in diameter. When 15 mm. is exceeded, dilatation is usually present. Dilatation of the pulmonary artery, when not a congenital abnormality, is usually an expression of the state of the pulmonary circulation. Moderate dilatation occurs in mitral stenosis without cardiac failure, particularly where it is the predominating lesion in a triostial stenosis. The dilatation noted in mitral stenosis may be functional or organic. For example, we have reviewed a series of cases of mitral stenosis without failure where roentgenologically there appeared to be definite dilatation of the major pulmonary vessels but in which at post mortem the dilatation if present did not exceed 2 mm. On the other hand, Assmann calls attention to distinct dilatation found at section in some cases, particularly where hypertension and arteriosclerosis of the lesser circulation is marked.

Increased width of the pulmonary artery occurs in congestive failure. The prominence of the pulmonic segment of the left cardiac contour is in proportion to the increased width of the hilar shadows. There is a diffuse clouding of the lungs, particularly at the bases. The exaggerated pulmonary markings occasionally have a nodular appearance. The degree of pulmonary artery dilatation and the clinical evidence of decompensation are often parallel but not necessarily so. Dilatation is apt to be most marked in cases which are severe and of long duration.

In the case of goiter, the prominent mid left cardiac contour seen in 30 to 40 per cent of cases, particularly in association with a long, narrow heart, is probably an expression of rotation rather than of true dilatation. Conceivably, however, a dynamic dilatation may be present and be related to the increased systolic output in hyperthyroidism. Dr. M. T. Woodruff and I reviewed a group of cases of toxic goiter before and after thyroidectomy. We found that, in general, six months after successful thyroidectomy, with the basal metabolism reduced to normal, the prominence in the left mid cardiac segment remained virtually unchanged.

Extensive occlusive processes in the smaller vessels may be found without any demonstrable roentgenographic abnormality. Dilatation, when it occurs, may be limited to the trunk or a major vessel. In chronic bronchopulmonary disease, such as bronchiectasis, emphysema or pneumonoconiosis, there is demonstrable evidence

of dilatation usually only when the condition involves most of the lungs and is of long standing; only occasionally, therefore, is there exaggeration of the pulmonic artery segment in these cases.

Most marked dilatation is seen in congenital heart disease, aneurysm and in sclerosis of the peripheral pulmonary vessels. The congenital lesions are being discussed by Dr. Roesler.

Ayerza's disease is a sclerosis of the pulmonary artery previously thought to be regularly luetic but which is now considered to be not necessarily specific. The main clinical feature is a marked cyanosis of the face, hence the descriptive term "black cardiacs" for the patients. The disease is said to be antedated by a chronic bronchitis which is persistent and progressive and associated with bronchiectasis. There are roentgen signs of emphysema, and tracts corresponding to the ramifications of the pulmonary artery are seen extending through the entire pulmonary area out to the periphery.

We have had two cases of "idiopathic pulmonary artery dilatation" where enlargement of all the pulmonary arteries, particularly of the main branches, was extremely marked and associated with marked pulsation. Aneurysm of the pulmonary artery is manifested as a localized dilatation occurring at the mid left cardiac contour situated anteriorly and pulsating.

"Hilum dance" (Pezzi) is the name given to a marked collapsing pulsation of the hilum most often seen with insufficiency of the pulmonic valve. The condition is rare but striking when seen. There is moderate dilatation of the pulmonic artery. This phenomenon may be found also in mitral stenosis with a relative insufficiency of the pulmonic valve. It is occasionally simulated in normal, long, narrow hearts of adolescent girls where the pulmonic artery segment is prominent.

The displacement of the pulmonary vessels by pulmonary and pleural disease must be also noted. Fibrosis with retraction of the upper lobe commonly elevates the hilar vessels.

COMMENT

Well-defined deviations from the normal in the size of the aorta yield evidence of disease. Our methods of measurement do not permit of precise determination of minor deviations, particularly in the ascending portion. Experience in roentgenoscopy of the chest, particularly as it permits observation of the variations in the great vessels due to the position of the diaphragm, of posture, of chest deformity and of age, has appeared more valuable than rigid application of mensuration. The smaller the deviation from normal the less valuable and sometimes more confusing is the information obtained. The roentgen examination of the aorta may not prove particularly valuable in the early diagnosis of luetic aortitis. Pulmonary artery dilatation, when not due to a congenital anomaly, is not characteristic of any particular disease but is rather an expression of the state of the pulmonary circulation.

It is my impression that as regards the two great vessels, if the roentgen findings are considered alone and completely apart from the clinical data, the value of the examination has been overestimated in the literature. In actual practice it has its definite place when considered as an integral part of the physical examination.

REFERENCES

1. Assmann, H. Die klinische Röntgendiagnostik der inneren Erkrankungen. F. C. W. Vogel, Leipzig, 1929.
2. Bordet, E., and Fischgold, H. Radiokymographie du coeur et des vaisseaux. Masson et Cie, Paris, 1937.
3. Canigiani, T. Zur Röntgendiagnostik des Aneurysma dissecans der Brustaorta. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1932, *45*, 416–420.
4. Hampton, A. O. Roentgenological measurements of the thoracic aorta. Modern Concepts of Cardiovascular Disease, July, 1938.
5. Hampton, A. O., and Jones, T. D. Clinical and roentgenological study of the aorta, with special reference to luetic aortitis. Am. J. Roentgenol. & Rad. Therapy, 1930, *23*, 390–395.
6. Pezzi, C. Radioscopic sign of the "hilum dance." In: Contributions to the Medical Sciences in Honor of Dr. Emanuel Libman. International Press, New York, 1932, Vol. 3, p. 931.

7. Stumpf, P., Weber, H. H., and Weltz, G. A. Röntgenkymographische Bewegungslehre inneren Organe. Georg Thieme, Leipzig, 1936.
8. Sussman, M. L., and Averbuck, S. H. Kymographic study of the ventricular pulsation in luetic heart disease. To be published.
9. Vaquez, H., and Bordet, E. Radiologie du coeur et des vaisseaux de la base. J. B. Baillière et fils, Paris, 1928.

DISCUSSION

Dr. George W. Holmes, Boston. As Dr. Sussman has stated, the cardiologist requires more assistance from the roentgenologist in the diagnosis of disease of the great vessels than in any other part of the heart examination. Dr. Sussman also rightly stresses the value of the fluoroscopic examination. An examination of the heart without fluoroscopy is of very little value. Films may be used to confirm the fluoroscopic studies, to make measurements from, when that is thought necessary, and occasionally they bring out unsuspected pathology either in the lungs or mediastinum.

Dr. Sussman emphasized and described the appearance of the aorta in older persons. Dr. Pfahler, I think, thirty years ago called attention to the distortion or the bulge of the aorta to the right and the prominence of the knob to the left and warned roentgenologists not to mistake that for aneurysm. In my observations as an examiner, there are quite a few men who have not found that out and for that reason I think it is worth while to bring up this warning once more.

I would also like to emphasize again the statement which Dr. Sussman made, that the measurements of the aorta during life do not necessarily conform to the measurements after death, and we must not be disappointed if the pathologist fails to confirm our diagnosis of a moderate dilatation of the aorta.

On the whole, I have enjoyed Dr. Sussman's paper particularly. I think it is an excellent presentation of the entire subject, and I want to thank the writers of all the papers for the opportunity of reading such a splendid symposium of the whole subject.

Dr. Samuel Levine, Boston. I was asked a question about angina pectoris, which reads: "What part does exercise play in length of life that a patient may enjoy with angina pectoris? Please discuss."

I do not know. We haven't very much knowledge concerning the rôle of exercise in relation to heart disease or in the production of heart disease or after it develops. My feeling about the matter is that the patient with angina pectoris should be allowed and even encouraged to do that exercise which he can do with comfort. That was the general teaching of MacKenzie. Sir Clifford Allbutt believed quite the opposite. He thought that the less they did for the rest of their lives, the longer they would live. Does exercise help the heart or does exercise harm the heart when it is diseased? I would say that theoretically exercise of the heart ought to be good. Life is not worth very much if you lie in bed for years and do nothing. I once made a remark that every time you lift your arm, you shorten your life, and it has become a boomerang now. I don't know if that is true. The heart has a certain amount of beats it is going to make and the sooner you get those beats made, the sooner it stops. You may save twenty-five thousand beats a day if you stay in bed, so we might stay in bed for the rest of our lives if we simply want to live, but as a practical matter, I think an intelligent or reasonable method of handling the patient is to permit him to do whatever he can without distress.

From a clinician's point of view, the roentgen examination of the aorta is of great value. Osler divided aneurysm into two groups: the aneurysm of sign and the aneurysm of symptoms. I think he omitted a third group, the aneurysm without signs or symptoms, the one you can see only with the roentgen ray.

I have seen luetic organic aneurysms as large as a grapefruit in which there were no physical signs or symptoms. The patient had pneumonia and he was going through a routine examination, with a routine roentgenogram of his lungs before going home, when this shadow was found. He had no signs or symptoms of heart disease or of any disease of the aorta. Before we knew that he had an aneurysm and after we knew where it was, we could find no signs of it by customary auscultation or physical examination. Therefore, I am certain that I must have overlooked aneurysm in the past and probably will in the future. The roentgen ray is of tremendous value to us in the diagnosis of aneurysm.

I was very sorry to hear that Dr. Sussman feels discouraged about making an early diagnosis of luetic aortitis. We would like very much to get roentgenologic help in this diagnosis. We cannot make, at least I cannot, an early diagnosis of luetic aortitis. It is suspected

if we find a positive Wassermann, but there are no signs that I know of that will tell you that luetic aortitis is present. In fact, luetic involvement of the aorta produces no physical disability until it does one of three things: produces an aneurysm and presses somewhere; closes the ostia of the coronary vessels, or produces aortic insufficiency. In other words, I do not believe that luetic aortitis causes any symptoms or signs merely by the inflammatory process of the aorta. It has to press on a neighboring structure. It has to close the coronary mouths or it has to produce an aortic leak before disability and symptoms result. Before these complications develop the condition would be symptomless and yet it would be in a stage where we would like to make an early diagnosis and institute therapy to prevent these other three sequelae from occurring.

I want to thank the Society very much indeed, first, for the opportunity of reading the papers in manuscript form before coming here, and secondly, for giving me the opportunity of discussing these papers and meeting you all here.

Dr. Hodges (closing). Three questions have been transmitted to me by the secretary. The first concerns itself with the triangular mass of fat which is sometimes visible at the cardiac apex. Presumably the questioner made mention of this because the presence of a fat deposit at this point is sometimes confusing when one attempts to determine the exact contour of the heart at the apex.

In all of the roentgenograms projected on the screen this afternoon, except those in which the heart border was completely hidden at one point or another, no sizable fat pad has been encountered which has seriously interfered with visibility of the cardiac apex. Dr. Holmes has pointed out that, if the fluoroscope is used, slight rotation of the patient will permit accurate differentiation between the cardiac contour and accessory extracardiac structures.

A second commentator very correctly points out that the diagrammatic representation of the heart as seen in left anterior oblique projection, shown by me on the screen, was at variance with the normal state of affairs. The left border of the heart in normal individuals should not overlap the spine if the angle of projection has been correctly determined in advance. The diagram shown was taken from the original left anterior oblique roentgenogram of a patient shown in the second diagram to have a 35 per cent increase in frontal plane silhouette area. With this degree of enlargement one expects partial superimposition of heart shadow and spine, and this is exactly what the diagram shows.

The third and last question deals with the character of pulsation to be observed along the right cardiac border and the actual identity of the chamber normally responsible for this portion of the silhouette. Your attention is called to the fact among the excellent anatomical specimens demonstrated by Dr. Roesler the right border of the heart distinctly represented the right auricle in each instance where direct frontal view was employed. However, in observing the pulsation of a normal heart at the fluoroscope one does not need to employ the kymographic method to identify ventricular pulsation in this quadrant. If, on the other hand, one examines a heart in which auricular fibrillation has been established, the totally irregular twitchings of the auricle become apparent between the irregular ventricular contractions. I believe that the more forceful transmitted contractions of the ventricle mask auricular movements under normal circumstances.

Dr. Steinberg (closing). Several questions have been asked regarding the method. We have an exhibit downstairs with photographs showing the apparatus and technique and we will be very glad to explain in detail how visualization is accomplished. Briefly, the method consists of two essential parts: the rapid injection of a radiopaque substance (diodrast) into the blood entering the cardiovascular structures and the taking of roentgenograms at the moment of opacification. Speed is accomplished by the use of a large bore needle-stopcock unit of 12-gauge. The needle is inserted into a large arm vein and its proper location tested by injection with normal saline. It is strapped into place and the patient is then ready for roentgenography. The dose of diodrast is 25 to 40 cc. of the 70 per cent solution. The Luer-Lok syringe with large bore tip which contains the diodrast is attached to the needle-stopcock unit and blood is gently aspirated. Being lighter, the blood rises to the top and during the injection carries the opaque material rapidly into the vena cava. The injection is made in two seconds or less. Average intervals between injection and exposure may be used

for visualization of the right side of the heart: for the superior vena cava, one and one-half second, and the right ventricle and pulmonary arterial tree, three seconds. Visualization of the left side is more variable (eight to twenty-three seconds) and requires a preliminary circulation time determination. We have used the sodium cyanide arm to carotid sinus method of Robb and Weiss and find that the onset of dyspnea is an accurate guide to the time of opacification of the left ventricle and aorta. Eventually, we hope that rapid serial roentgenography will provide a picture of each division of the thoracic cardiovascular system with one injection.

In regard to Dr. Levine's question about contraindications, the usual contraindications for diodrast listed by "New and Nonofficial Remedies" are applicable save that we have used it in tuberculosis without ill effects. Diodrast is a very stable iodine compound which is rapidly eliminated. We have carefully studied the pharmacology of diodrast and by continuous electrocardiographic tracings during injection in normal as well as in patients with angina pectoris found no ill effects. There is routinely an elevation of the pulse rate and drop of the blood pressure which is usually transitory in nature. The drug is a vasodilator of the coronary circulation. More of the pharmacology will appear in the method paper.*

Dr. Freedman (closing). I have been asked by Dr. Ross Golden as to how frequently we have found adhesions between the pericardium and anterior chest wall. I do not remember the exact figures but would say offhand that the pericardium is adherent to the sternum in about 40 per cent of the cases.

As our experience with this condition has increased, we have come to realize that the adhesions by themselves, as Dr. Levine pointed out, are of secondary importance. The finding of a rigid pericardium as demonstrated by the inspiratory and expiratory films and by the relatively small heart shadow are of primary importance.

Dr. Levine made the statement that whenever he looks for cardiac compression he expects to find a small heart shadow. This is generally true, as the dimensions and measurements of these hearts are small. However, occasionally one may find a cardiac shadow which is quite large, due either to an underlying and preexisting cardiac pathologic process or to excessive thickening of the pericardium.

I also would like to discuss another statement made by Dr. Levine concerning the calcification of the pericardium. He said that the finding of calcified plaques in the pericardium does not necessarily mean that it causes symptoms. In our material, however, in each instance where there was calcification of the pericardium, the patients had clinical symptoms of cardiac compression and were operated upon by Dr. Beck.

Dr. Levene (closing). Both Dr. Holmes and Dr. Levine asked a question and I will direct the answer mostly to Dr. Levine because I want to put it in the form of a rhetorical question.

Why is it that the clinician is so bold as to make a diagnosis when we make a negative diagnosis by roentgen ray? We all know of many cases where the clinician will tell a patient, "No, there's nothing wrong with your heart; you go out and play 18 holes of golf," and sometimes he gets away with it and sometimes he doesn't.

We have 10 cases now in which that happened. The clinician with his electrocardiograph called it a negative heart. We called it coronary artery disease and necropsy showed that we were right.

The next point is on the question of a finding of angina pectoris. I, not being a cardiologist, am probably not qualified to find fault with that term, but I don't like it. You either have pain in the heart or you have coronary artery disease. The pain in the heart which accompanies coronary artery disease, it seems to me, would much better be called coronary artery disease, not angina pectoris, which is just a symptom of pain in the chest. And, of course, it is our feeling that where you do have coronary artery disease, you see real structural changes in the heart.

The next point is the question, do you get a flat ventricle in other conditions? Yes, Some of the previous speakers showed films that apparently had flat ventricles, and you see it in constrictive pericarditis, thyrotoxicosis, and myocardial impairment. However, when in addition to these changes in contour, you see a localized diminution in the force of the beat, we feel that that localized diminution means localized myocardial damage—and nothing will better explain local myocardial damage than coronary artery disease.

*Am. J. Roentgenol. & Rad. Therapy, 1939, *41*, 1–18.

The question of the physiology in arteriovenous aneurysm has been brought up. The systemic, the peripheral, pressure is low when the shunt of blood is from the aorta to the pulmonary artery. However, as shown by Emile Holman some time ago, when the shunt is the other way, from the pulmonary artery or from the vena cava to the aorta, the pressure is high.

The next question pertains to the contraction of the left ventricle and hypertension. Is the force of beat increased? We believe it is. Why doesn't the clinician hear a forceful beat? Well, there are two explanations for that. First, many of these cases with a systolic pressure of 240 are obese, and in these people the sounds are diminished. Secondly, many of these cases with hypertension have myocardial damage and in myocardial damage the force of beat is diminished.

The next question is by Dr. Fineman of New York City. How do you determine the precise location of the apex, particularly when elevated, as the thickness of the wall as measured on the film depends on the exact location of this point? That is a question that is asked very often. I think confusion is due to the fact that we are uncertain as to what we are looking for. What is the apex and why do we call it the apex?

The heart is considered to be a pyramid, and geometrically the apex of a pyramid is the thinnest point opposite the base. If you look for the thinnest, the narrowest part of the heart, and place that point wherever you find it, above the diaphragm or below (sometimes you will see it in the gas bubble of the stomach as in aortic valvular disease), that is the apex, and connecting that line to the auricular-ventricular junction gives you an approximate idea of the thickness of the wall.

This thickness of the wall, as we measure it, does not coincide exactly with what the pathologist will find. We have just reviewed, and the day before I came here we had our last figures, 50 cases which we measured and which the pathologist measured. There is an average discrepancy of 2 millimeters in the thickness of the wall as we measured it. It is not a question of exactly how thick it is, because after all, you cannot make a medical diagnosis by mathematics, but where the wall is thick, you get a fairly good idea by this measurement.

Vol. 42, No. 1

PERSISTENT VERTEBRAL PROCESS EPIPHYSES*

By WILBUR BAILEY, M.D.
LOS ANGELES, CALIFORNIA

SECONDARY centers of ossification may appear opposite the tips of any of the major vertebral processes. The transverse processes show such centers with regularity during the growth period; they are not rare at the tips of the inferior articular processes, and they occur at the tips of the superior articular and spinous processes.

During the growth period these centers of ossification are usually multiple or bilateral and are not likely to be misinterpreted. However, if fusion fails to occur at the normal time a "persistent epiphysis" may result and the ossicle which remains opposite the process is more than a pathological curiosity, for it may be mistaken for a fracture. These anomalous ossicles have been described by various authors as opposite different vertebral processes and have been considered as anomalous bones or "accessory articular processes" in the case of the inferior articular processes (Rendich and Westing[9]), or as epiphyses. It seems logical that these centers of ossification should be grouped together and considered as secondary epiphyses, for in other bones the phenomenon of epiphyses which appear toward the end of the growth period, but which are not a constant finding, is well recognized.

TYPES OF PERSISTENT EPIPHYSES

Graberger[5] finds in 0.7 per cent of all spines a persistent epiphysis opposite the transverse process of the first thoracic vertebral body. In Figure 2 such an ossicle is seen. Such ossicles are described as being frequent in the lumbar region where, according to Janker,[6] they may be mistaken for stones in the renal pelvis. In the literature and according to this author, "persistent epiphyses" may occur opposite the transverse processes of the vertebrae of any section of the spine. Rudimentary ribs are particularly frequent opposite the transverse processes of the first lumbar vertebra,

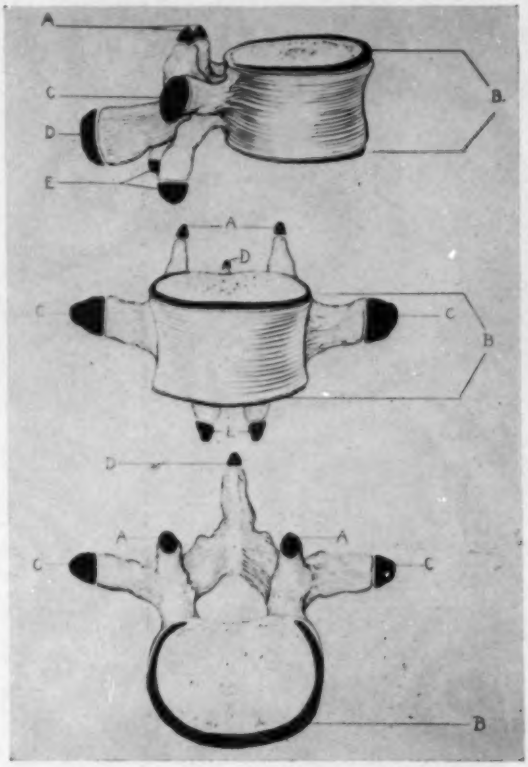


FIG. 1. Diagrams of a vertebra showing the points at which persistent epiphyses may occur. *A*, superior articular processes; *B*, ring-like vertebral border epiphyses; *C*, transverse processes; *D*, inferior articular process; *E*, spinous process.

but may occur with any of the lumbar vertebrae, and the ossicle so formed may not be distinguishable from a persistent epiphysis of the transverse process.

The spinous processes may also have small independent centers of ossification persisting opposite their tips in later life (Disse[3]). This phenomenon occurs particularly often at the region of the transition of the lumbar into the sacral spine. The ap-

* From the Department of Radiology, University of Southern California School of Medicine, Los Angeles, Calif.

pearance of these ossicles is rather peculiar when they are present in cases in which there is also a failure of fusion of the spinous processes (spina bifida occulta). Under these circumstances the gaping split is seen between the ununited spinous processes in the center of which a small piece of bone about the size of the tip of a spinous process is seen to be present entirely separate from any of the surrounding bones. This ossicle is well shown in Figure 3 and merely represents the secondary center of ossification which, despite the failure of fusion of the underlying spinous process, has developed in its normal place. This appearance is particularly striking when the spina bifida de-

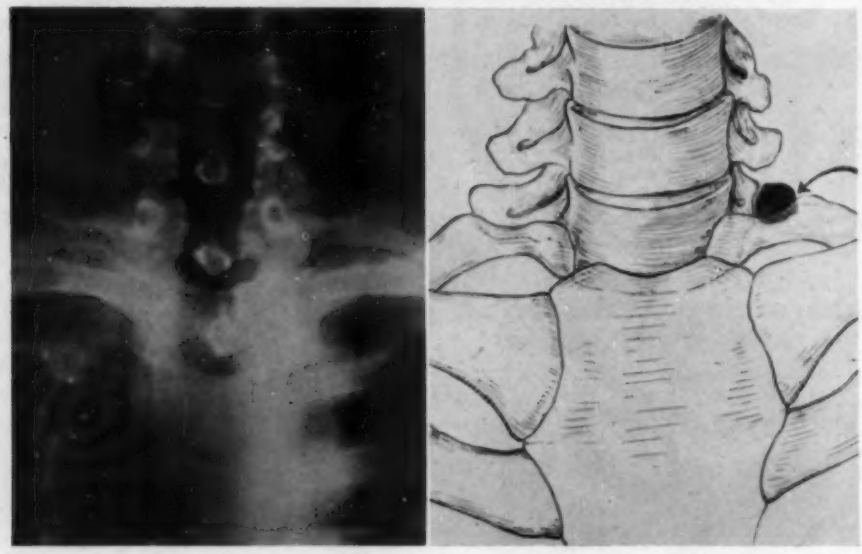


Fig. 2. Persistent epiphysis opposite the transverse process of the first thoracic vertebra.

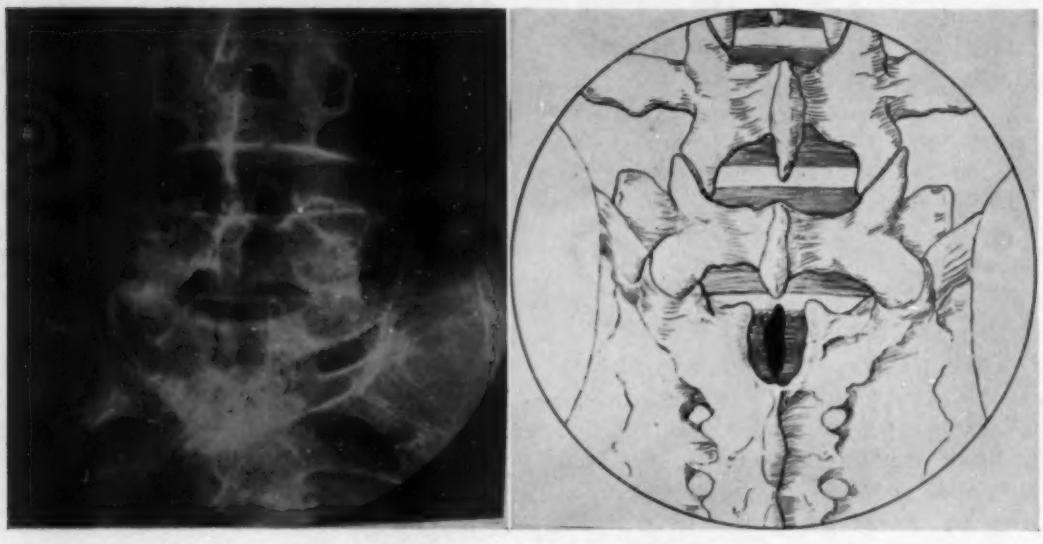


Fig. 3. Spina bifida occulta defect of the first sacral segment. The secondary epiphysis has developed nevertheless and can be seen in the gap but not attached by bone to the bifid spinous process.

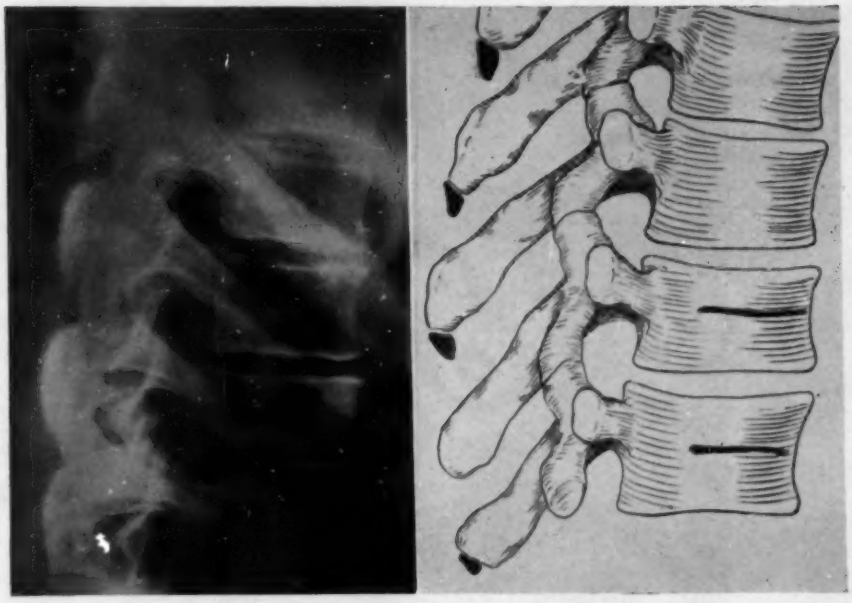


Fig. 4. Persistent epiphyses opposite the spines of the fifth, sixth and eighth dorsal vertebrae. (Courtesy of Dr. Richard T. Taylor.)

fect involves a number of the lumbar vertebrae. The secondary epiphyses can then be seen (Schmorl[10]) as a chain of linear small bones suspended in the middle of the gap between the bifid spines. When an epiphysis persists opposite the spine of the seventh cervical vertebra, fracture is likely to be wrongly suspected. In Figure 4 a number of such ossicles are seen in the thoracic region which, because of their multiplicity alone (as well as the fact that there was no history of sufficient trauma in any of these cases), would not likely be interpreted for anything but anomalies.

Centers of ossification opposite the tips of the inferior articular processes of the lumbar vertebrae are not especially rare. In a previous communication,[1] at which time the entire literature on the subject was reviewed, it was pointed out that these ossicles have been found opposite the inferior articular processes of any of the lumbar vertebrae. In the 19 cases which were reported in the literature, 6 were bilateral although only 1 of my 9 cases showed bilaterality. Partial fusion between the inferior articular process and the adjacent

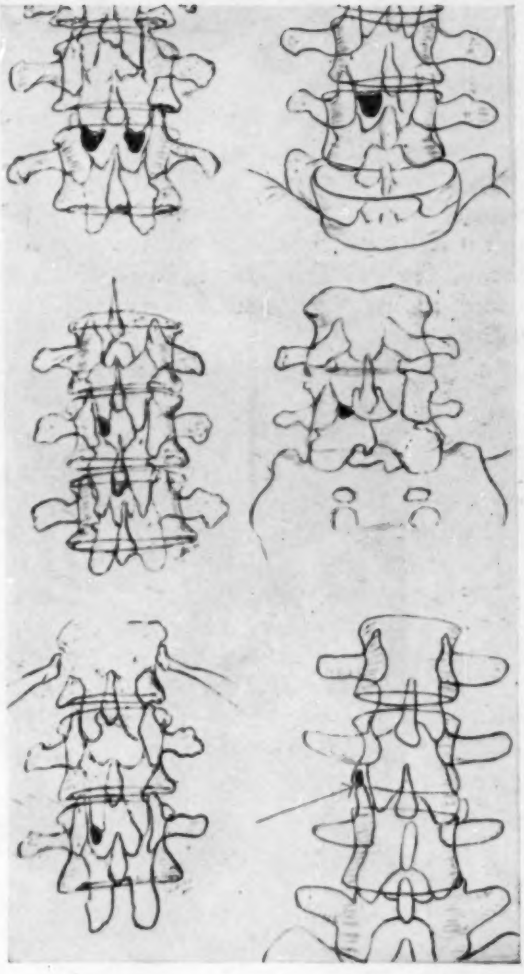


Fig. 5. Diagrams typical of persistent epiphyses of the inferior and superior processes.

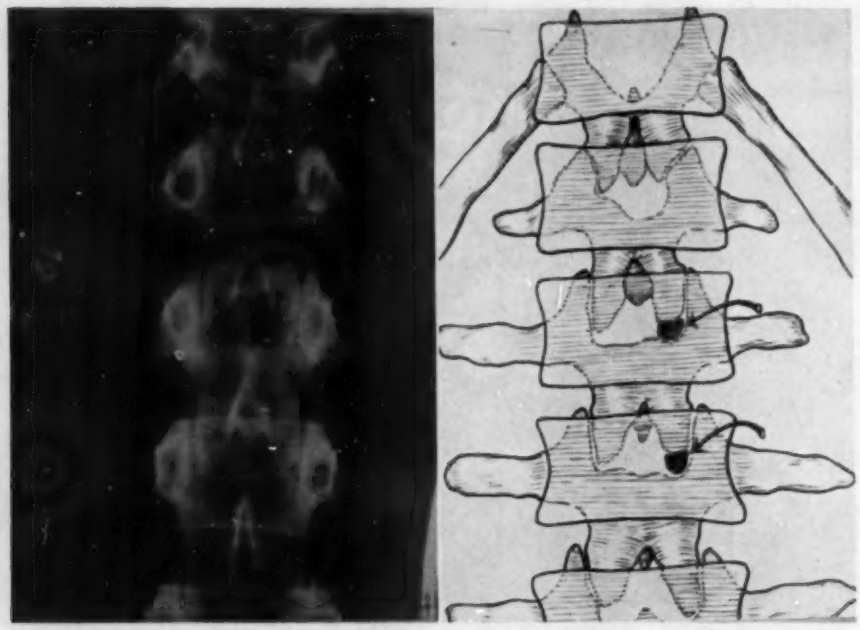


Fig. 6. Persistent epiphyses at the inferior processes of the left first and second lumbar vertebrae.

ossicle may be present so that a mesial notch in the inferior articular process may be the only evidence of an anomaly. Diagrams of a number of such anomalies appear in Figure 5, and Figure 6 shows the left inferior processes of both the first and second lumbar vertebrae to be so affected. More recently Farmer[4] has reported 40 cases of articular process anomalies in the lumbar spine in which he finds the incidence to be 1.5 per cent. He wisely states further: "Time no doubt will show this incidence to be low, since more and more of these smaller bones will be discovered as technique is improved and attention is focussed on these anomalies."

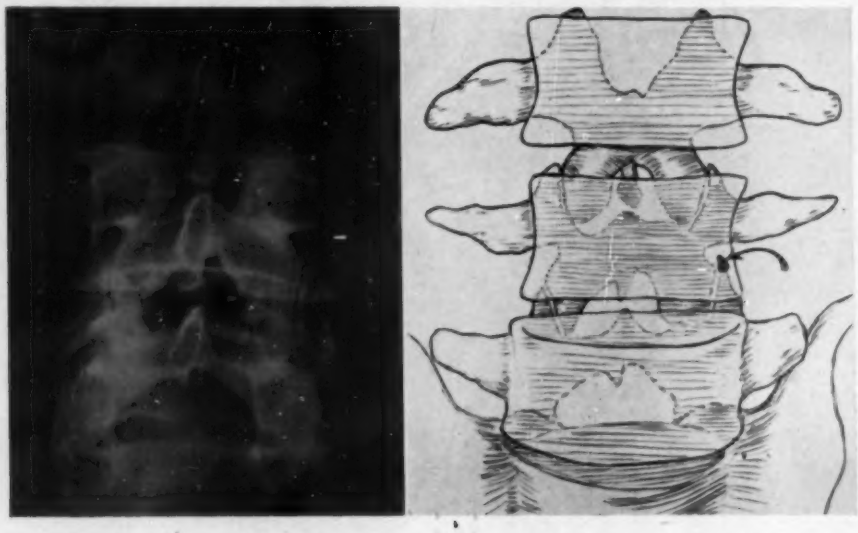


Fig. 7. Persistent epiphysis opposite the left superior articular process of the fifth lumbar vertebra.

Superior articular process anomalies are much rarer than those in the inferior processes. Five have been described in the literature and a sixth is shown in Figure 7.

DEVELOPMENT

The very early morphogenesis of the vertebrae is not within the scope of this paper. Jordan and Kindred[7] point out that shortly after the beginning of differentiation of the embryonal vertebra, three centers of chondrification appear on each side. One center is in each neural process, or the neural arch, one in each side of the vertebral body, and one in each costal process. By a continuous growth and fusion of the several parts a cartilaginous vertebra is formed. The transverse and articular processes grow out from the side of each of the neural arches.

The ossification of each vertebra occurs later and starts in three centers: one in the body and one in each half of the neural arch. Ossification starts at eight weeks and continues until after birth. A cartilaginous plate is formed at the ends of the bodies of each vertebra which plays an important part in protecting the integrity of the cancellous bone of the body throughout life.[2] Of importance in this discussion is the fact that a ring-like secondary center of ossification develops at the periphery of these cartilaginous plates at about the seventeenth year and usually fuses with the bodies of the vertebrae at about the twentieth year. These rings are shown in Figure 1 at *B*. Should a failure of fusion occur between these structures and the adjacent vertebral body, they will be seen in profile in the lateral roentgenograms as a small triangular piece of bone, opposite the ventral corners of the vertebral bodies. The vertebral bodies, therefore, may have persistent epiphyses at their rims just as there may be persistent epiphyses opposite the tips of vertebral processes.

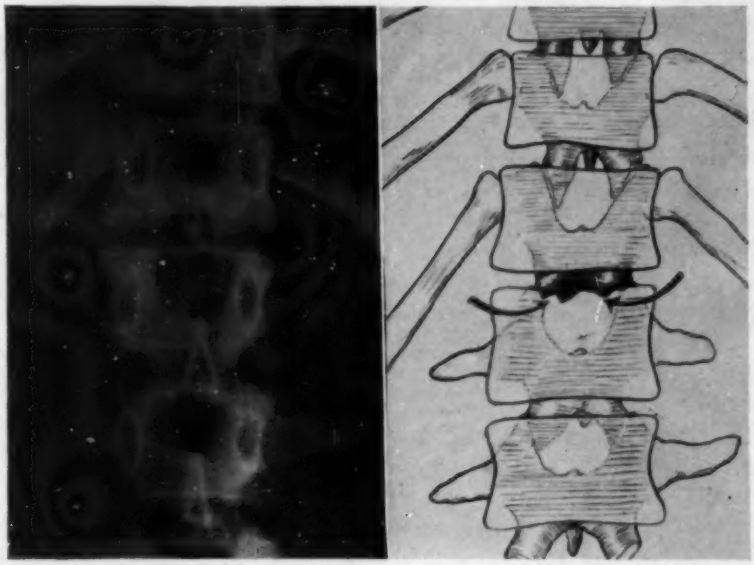


Fig. 8. Rudimentary inferior articular processes of the twelfth thoracic vertebra. These processes are scarcely long enough to articulate with the superior processes of the vertebra below.

In the literature the formation of the vertebral processes and their origin from the primitive neural arches is adequately explained. Regarding persistent epiphyses, however, no description of anatomical specimens can be found. It is not likely that much knowledge would be gained, how-

ever, in this particular situation over that which is already presented by the roentgenograms.

ABSENCE OF THE INFERIOR ARTICULAR PROCESSES

Mueller[8] has described a case in which there was a complete absence of the inferior articular processes of the second lumbar vertebra. In view of the embryological origin it is not surprising that sometimes unusually long articular processes sprout forth (opposite which there is apt to be a persistent epiphysis) while sometimes, on the contrary, they are very rudimentary, as in Figure 8, or completely absent. We have noted the twelfth thoracic vertebra to have very rudimentary processes fairly often, although the adjoining vertebrae were not so affected.

COMMENT

The great importance of all these persistent epiphyses is the danger that they may be mistaken for fracture. There are, however, a number of differential points.

(1) Such persistent epiphyses in the inferior articular processes are not very unusual, while isolated fractures in this region are a rarity. The history of antecedent trauma in the anomaly cases may be vague or entirely absent as it was in half the cases of a previous series.[1] On the contrary, the pain following the cases of fracture has proved to be prompt, severe, and disabling.

(2) Because of ligamentary pull, a fracture fragment is usually displaced from its base, and is therefore not likely to be seen in close apposition with the opposing bone cortices completely parallel as is the case with the anomalies.

(3) Persistent epiphyses are often multiple or bilateral. Multiple fractures may occur but lack regularity.

(4) In the case of the spinous and transverse processes, at least, fractures usually occur near the base and not at the tips.

(5) Recent fractures are not likely to be mistaken, for a typical fracture line is irregular and serrated and there may be small comminuted fragments present. The persistent epiphyses, however, are smooth, well rounded, and have a definite cortex which is parallel to the cortex of the adjacent processes.

SUMMARY

(1) Separate centers of ossification may be present opposite the tips of any of the vertebral processes. If union between these secondary ossification centers and the adjacent process fails to occur at the end of the growth period, the resulting ossicle is most logically designated "a persistent epiphysis."

(2) The chief clinical importance of these persistent epiphyses lies in the fact that they may be mistaken for fractures. Differentiation is nearly always possible, however.

REFERENCES

1. Bailey, W. Anomalies and fractures of the vertebral articular processes. *J. Am. M. Ass.*, 1937, *108*, 266–270.
2. Bailey, W., and Taylor, R. T. Importance of lesions of intervertebral discs in relation to trauma. *California & West. Med.*, 1936, *44*, 509–513.
3. Disse, J. Quoted by Schmorl and Junghanns, ref. 10.
4. Farmer, H. L. Accessory articular processes in the lumbar spine. Am. J. Roentgenol. & Rad. Therapy, 1936, *36*, 763–767.
5. Graberger, G. Quoted by Schmorl and Junghanns, ref. 10.
6. Janker, R. Persistierende Apophysen der Querfortsätze der Wirbelsäule, des Beckenkamms und des Trochanter minor? *Rontgenpraxis*, 1930, *2*, 501–505.
7. Jordan, H. E., and Kindred, J. E. A Textbook of Embryology. Second edition. D. Appleton & Co., New York, 1932.
8. Mueller, W. Ueber eine bemerkenswerte Form von Wirbelsäulenmissbildung. (Völliges Fehlen von Gelenkfortsätzen.) *München. med. Wchnschr.*, 1932, *79*, 356.
9. Rendich, R. A., and Westing, S. W. Accessory articular process of the lumbar vertebrae and its differentiation from fracture. Am. J. Roentgenol. & Rad. Therapy, 1933, *29*, 156–160.
10. Schmorl, G., and Junghanns, H. Die gesunde und kranke Wirbelsäule im Röntgenbild. Georg Thieme, Leipzig, 1932.

Vol. 42, No. 1

MOTION IN THE VERTEBRAL COLUMN*

By JOSEPH F. ELWARD, M.D.
WASHINGTON, D.C.

THE purpose of the present communication is to synthesize currently accepted and established facts relating to motion in the vertebral column, particularly in its dorsal and lumbar portions, to consider anew all the factors involved therein, and to illustrate by means of roentgenograms of living subjects the range of mobility in the vertebrae.

For centuries the study of the comparative degree of mobility of the several component segments of the vertebral column has enlisted the attention and interest, not only of anatomists and orthopedists, but of mathematicians and philosophers as well. Indeed, Andreas Vesalius, sometimes called the father of anatomy, in his admirable work on the structure of the human body which bears the date 1543 A.D., presented a minutely detailed description of the vertebral apophyses, with particular reference to their varying directions, and even illustrated the difference between the latter by means of two lines, one oblique, the other vertical.

Among post-Vesalian anatomists, Jakob Benignus Winslow, a Danish professor, whose textbook entitled "Exposition anatomique" was next to Vesalius' work the authoritative source of reference for nearly a hundred years, in 1730 published probably the earliest definite contribution to the subject of motion in the vertebral column in the form of a meticulous description of the apophyseal facets of the vertebrae, with a discussion of their respective reciprocal intercontact during movements of the head, neck, and the rest of the spine.

Following Winslow, in 1827 Ernst Heinrich Weber executed perhaps the first actual measurements of movements of the human vertebral column on cadavers, two male and one female, as well as living subjects, young men aged from twenty to thirty-two years. Weber found that the cervical are the most mobile vertebrae, while between the dorsal and lumbar vertebrae the movement of rotation and inclination is so distributed that the lumbar vertebrae can be flexed only anteriorly, posteriorly, and laterally, but practically not at all about the longitudinal axis of the vertebral column, and the majority of dorsal vertebrae cannot be flexed but only rotated.

In 1859, Bishop, an English geometrician and physician, demonstrated before the Medical Society of London the mechanism of the spinal column, the structure of the intervertebral cartilages, and the manner in which these confer flexibility on the cervical, dorsal, and lumbar regions. The purpose of this exposition was to promote knowledge of causes and treatment of lateral curvature of the spine.

The German physiologist, Volkmann, apparently unaware of the previous work of Weber, in 1872 reported exhaustive measurements of rotatory movements of the vertebral column on dead and living subjects, his figures differing widely from those recorded by Weber, and thus serving to involve the data of both investigators in controversy.

A French savant, Guérin, in 1877 devoted a lengthy memoir to a study of movements of inclination and flexion of the vertebral column, in which he revealed the existence of four centers of lateral inclination (atlanto-occipital, cervicodorsal, dorsolumbar, and lumbosacral), and described in detail their relation to articular and muscular systems during these movements.

Two years later, in 1879, Morris, an English writer, pointed out that nowhere in the lumbar vertebrae are the inferior processes of one bone in close juxtaposition to the superior processes of the next, and that the facets on the two sides can in no instance be placed in immediate apposition

* Read at the Fifth International Congress of Radiology, Chicago, Ill., Sept. 13–17, 1937.

at the same time, thus rendering rotatory movements in the lumbar region possible solely by virtue of this space between articular facets.

In 1890, Löhr in Germany evolved presumably the first systematic method for the mensuration of forward and backward movements of the spine in living subjects, whereby measurements were made from shadows projected upon a screen, and the degree of angularity determined in 47 subjects ranging in age from seven to fifty-seven years.

Hughes of Edinburgh in 1892 published results of collective investigations of rotary movements of the human vertebral column. In a series of measurements which he himself conducted, Hughes sought to reconcile discrepancies in figures previously submitted in turn by Weber and Volkmann.

In 1896, Hall, an American author, exhibited a number of tracings which purported to represent with a tolerable degree of accuracy the amount and kind of possible mobility in the spine of a child in dorsal decubitus. The extraordinary extent of mobility thus demonstrated, in his opinion, the necessity for improved fixation in bed-treatment of tuberculosis of the spine (Pott's disease).

A summary of his results from the study of normal spinal movements in relation to scoliosis was, in 1900, made public by Lovett, who asserted that torsion and side flexion of the spine are parts of one combined movement, neither existing to any extent individually, lateral deviation of any part of the spinal column being therefore necessarily associated with torsion (rotation) at the site of the deviation. Moreover, Lovett stated, in flexed positions side bending is associated with torsion in one direction, in extended positions by torsion in the opposite direction; and, further, that from the kind of torsion observed in scoliosis, obviously the deformity originated in the flexed position of the spine, the correction of rotation therefore being apparently effected logically by throwing the spine into extended positions and in taking side bendings from extended positions.

In 1906, Keene, of the Harvard Medical School, described his experiments on mechanical rotation of the normal spine, in which he found that rotation of vertebrae may be produced when the extremities of the spine are fixed by pressure on any of the intermediate ribs, the vertebrae articulating with ribs on which pressure is applied generally exhibiting maximum rotation, and distortion of ribs occurring approximately in proportion to the amount of force employed following neutralization of the initial resistance of the rib, the most effective points of pressure and counterpressure being upon corresponding ribs as far as possible from the midline anteriorly and posteriorly.

Three years afterward, in 1909, Hans Virchow contributed the first of his studies on sagittal-flexor movements in the human spine, this initial monograph dealing primarily with the atlanto-axial articulation, the degree and range of motion in which Virchow represented by geometrical diagrams in conjunction with the demonstration of an anatomical preparation.

Engelhard, in 1910, described a simple mensural apparatus of his own design for the estimation of curvatures of the spine as applied in the examination of some 70 children aged from six to fourteen years, results in 15 of whom he illustrated by means of diagrammatic sketches and accompanying tables of figures, all intended to demonstrate position, form, and mobility in the sagittal plane.

In 1911, Novogrodsky, in his thesis for the degree of doctor of medicine at the University of Bern, reported data derived from the measurement of four preparations of vertebrae, the figures recorded by him being, of course, too few to serve as a basis for definite conclusions.

Blumenthal, in 1912, in his turn described an easy method for the representation and estimation of bodily movements, especially of the spinal column, consisting

of the attachment to the body of the subject along the line of the spinous process of small circular pieces of adhesive plaster through which linen threads are run, these threads being arranged in parallel fashion one under the other over smooth surfaces and held taut by suspended leaden weights, each thread bearing at an equal distance from the adhesive plaster disks a marker in the form of a securely attached black bead, these beads indicating in their entirety the superficial curves of the vertebral column and following closely all anterior and posterior flexions, the body being enabled to move freely without the restraint imposed by instruments of mensuration. The course pursued by these bead-markers and corresponding to various positions is delineated by means of varicolored crayons upon a suitably placed ground glass plate. The successful application of the method is illustrated by a number of diagrams.

In 1919 Hans Virchow published the second of his contributions to the subject in the form of a brief comparative review of anatomical and roentgenologic findings involved in sagittal flexor movements of the human vertebral column, this being apparently the earliest reference in the literature to the employment of roentgen rays for the study of motion in the human spine.

Bradford, in 1922, presented descriptions with illustrations of the various devices ordinarily employed to trace variations in the spinal lines, and discussed the several factors involved in the causation of distortions of rotation in the trunk.

One year later, in 1923, Kellogg reported the results of experiments designed to test the strength of the spinal symphyses. Pressures of 1,195 and 800 pounds respectively were applied to the unsupported anterior and posterior surfaces of the body of the second lumbar vertebra, the neural arch being crushed under the first, and the body itself crushed to bits under the second stress; while following the application of 1,315 pounds at the side of the tenth thoracic vertebra, the body was crushed without materially impairing the articulation with the adjacent cartilages, these tests demonstrating that in the spinal segment involved therein, the strength of the vertebral symphyses was greater than that of the bodies of the vertebrae.

Probably the first serious attempt at clinical application of accumulated knowledge concerning the mobility of the human spine was made in 1924 by Keller. He attributed the relative amount and extent of motility of the several segments of the spinal column to the size and thickness of the respective intervertebral cartilages.

In the same year, Cyriax, a London physician, demonstrated a simple inexpensive apparatus for measurement of the amount of malrotation, called by him the "spinal torsionometer." The device consists of an aluminum block 4 by 2¾ inches in size, with a scale for recording degrees, above which there is suspended a fine needle which registers immediately the angle at which the block is lying. The subject of examination is placed face downwards on a couch, a dermographic line is drawn through the tips of the spinous processes in the region desired, with transverse lines to denote the middle of each such process. The torsionometer is placed transversely on the points of intersection of the line on successive vertebrae and the amount of rotation (if any) read off. If placed longitudinally over each point of intersection, the amount of anteroposterior curve can also be estimated.

In 1929 Heuer published the first of his studies on anterior and posterior flexion of normal vertebrae, with particular reference to modifications in range of movement sustained in individual vertebral columns in kyphosis and lordosis. This was followed in 1930 by a second contribution by Heuer on rotation of the vertebral column, both physiologic and scoliotic, in which he asserted that there is no connection whatever between the two types of rotation and that the latter therefore cannot be derived from the former.

In that same year (1929), Dittmar, an

orthopedist of Heidelberg, presented an account of his roentgenological observations on articular processes of vertebrae during sagittal and lateral flexion, his contribution apparently representing the first earnest effort to utilize roentgen rays for the investigation of the mechanics of motion in the spine.

For the study of movement involved in sagittal flexion Dittmar, using himself as a subject, made roentgen studies in three positions, representing respectively the terminal position of extension, the terminal position of flexion, and the relaxed neutral, median, or erect position. By means of these he demonstrated that flexion of the lumbar portion of the spine from the erect position is greater than extension, and that extension is greatest between the sacrum and fifth lumbar as well as between fifth and fourth lumbar vertebrae. The medial point of sagittal movements between two lumbar vertebrae was found to lie in the nucleus pulposus. Movements were shown to occur not around a single axis, but about several variously situated transitory axes. Notable shearing was remarked in the case of the lumbar vertebrae during sagittal flexion. Elevation of the intervertebral discs anteriorly was not observed to correspond to their decline posteriorly, and vice versa.

Two male subjects, aged twenty-two and twenty-four, with normal spines were utilized for the observation of lateral flexion. Roentgenographic studies were made in right and left lateral inclination, and median erect positions respectively. It was shown that mobility of the lumbar portion of the spine is in respect to lateral flexion greatest between the third and fourth and the fourth and fifth lumbar vertebrae. During lateral flexion the fifth lumbar vertebra was seen to rotate around a sagittal axis in a direction opposite to the direction of lateral curvature. Here also the medial point of movements of lateral flexion between two lumbar vertebrae was found to lie in the nucleus pulposus. The roentgenograms demonstrated further that movements between first and second, second and third, and third and fourth lumbar vertebrae occur around one and the same axis, while between fourth and fifth lumbar as well as between fifth lumbar and first sacral segment movements take place around variously situated transitory axes, whose deviation one from another was marked in the case of the lumbosacral, and barely perceptible in the case of the fourth-fifth lumbar vertebrae. Except for the fifth lumbar vertebra no shearing was noted. The unilateral elevation of the intervertebral discs which was observed corresponded to the compression on the opposite side, and vice versa.

More recently, in 1934, Wiles discussed the practical advantages of vertical roentgenography with the Potter-Bucky diaphragm in the routine examination of spinal deformities, and demonstrated the application of the method in normal subjects and in acrobats. One of the latter could hyperextend the dorsal spine to a remarkable degree with at least 90 degrees of rotation spread over the dorsal and lumbar spines. Wiles agrees with other observers that in addition to intervertebral movements of rotation, lateral bending, flexion, and extension, there is during flexion slight forward displacement of each vertebra on the one below it, and during extension a slight backward displacement, these bodily displacements of the vertebrae exerting a shearing strain on the intervertebral discs, and probably playing an important part in the production of radial tears in the discs and injuries to the cartilaginous plates as described by Schmorl, and hence in nuclear prolapse.

According to Lewis, who expresses the consensus of the majority of anatomists, permissible intervertebral movements comprise in their entirety flexion, extension, lateral motion, circumduction, and rotation. The comparative extent and freedom of these movements as related to the respective segments of the vertebral column may, Lewis explains, be stated briefly as follows: Flexion, the most extensive of all

the movements, is freest in the lumbar region; extension, limited by the anterior longitudinal ligaments and the approximation of the spinous processes, is freest in the cervical region; lateral movements, possible in any part of the column; circumduction, a succession of movements previously enumerated, is quite limited; rotation, produced by twisting of the intervertebral fibrocartilages, occurs to a slight extent in the cervical region, is free in the upper part of the thoracic region, and is absent in the lumbar region.

In general, the extent of motion is limited at all points by the common vertebral ligaments, although it depends in part upon the width of the bodies of the vertebrae and the depth of the intervertebral discs. For example, in the lumbar region where the bodies are large and wide and the discs very thick, free motion is possible, though limited; while in the cervical region, although the discs are thinner, the bodies are smaller, consequently the maximum freedom of motion is permitted.

Flexion is produced by the sternomastoid, longus capitis, longus colli, scalenus anterior, rectus abdominis, and psoas major muscles, when acting with their fellows on the opposite side; extension, by the intrinsic muscles of the back, aided in the neck by the splenius, semispinalis dorsi and cervicis, and the multifidus; lateral motion, by the intrinsic muscles of the back, the splenius cervicis, the scalenii, quadratus lumborum, and the psoas major, these inclining the column to their own side only; and rotation by the unilateral action of the sternocleidomastoid, the longus capitis, the scaleni, the multifidus, the semispinalis capitis, and the abdominal muscles.

Brailsford dissents from the general opinion relative to extent and freedom of motion as expressed by Lewis. In fact, during the preparation of a film intended to facilitate the study of movements of the vertebral column by means of roentgenogram and cinema in female acrobats, Brailsford, Lockhart and Fowler found that extension is greater than flexion and that the maximum range of movement occurs in the lumbar segment, and the minimum in the dorsal. These phenomena were demonstrated on a girl who was able to bend backward and place her head between her thighs. Brailsford further insists that standard anatomical texts are erroneous not only with respect to statements concerning extension and flexion but also to those relating to rotation in the lumbar region, and asserts that the latter movement is permitted to a greater extent than has previously been thought possible.

Wilson and Cochrane declare that the widest range of movement is in the cervical spine, the occipito-atlantal joints permitting motion only in the anteroposterior plane, as in nodding of the head, rotation of the latter being effected by the atlantoaxoid articulations. They state that the midportion of the cervical spine permits lateral bending (approximation of ear to shoulder). According to these writers, flexion and extension appear as principal movements in the lower cervical region. The thoracic region in its turn permits movements of rotation, flexion, and extension, also a slight degree of lateral bending. All thoracic movements are said to be limited by interference from the ribs and the sternum, the actual movement between any two vertebrae being slight, but the aggregate result considerable. Continuing their exposition, Wilson and Cochrane explain that the lumbar region permits definite movements in the anteroposterior and lateral planes, while rotatory movements are possible only with difficulty owing to the crescentiform articular facets. They assert that motion is most marked at the joints representing the junction of the fixed and movable portions of the spine i.e., the dorsolumbar and lumbosacral articulations.

Davis states that flexion and extension are free in the cervical and lumbar regions, while rotation is slight in the cervical region, free in the upper portion of the dorsal, and gradually diminishes and ceases in the lumbar region.

Fig. 1. Lumbosacral region. Erect posture (retouched).

The study of a young female (aged sixteen) with more than the usual range of motion in the vertebral column is of considerable interest. Roentgenograms of the lumbosacral and dorsolumbar regions were made in the erect posture and in the extremes of flexion and extension. The most striking observation in this study is the rôle of the hip joints in flexion.

Figure 1 shows the subject in the erect posture. The normal lumbosacral articulation shows the inferior aspect of the fifth lumbar almost paralleling the superior aspect of the first sacral segment, the lumbosacral space being 0.30 cm. wider at the anterior lip, the precise figures being 1.10 cm. anterior and 0.80 cm. posterior. The fourth lumbar space shows a variation of 0.30, the third lumbar 0.370, and the second lumbar 0.475, the anterior aspects being the wider.

Figure 2 shows the lumbosacral region in flexion. The lumbosacral space shows a variation of 0.15 cm., the anterior aspect being wider. The fourth and third lumbar spaces each show a variation of 0.15 cm., the posterior aspect in each instance being the wider. The second lumbar shows no variation, the adjacent surfaces of the second and third lumbar vertebrae being parallel.

Figure 3 shows the lumbosacral region in extension. The fifth lumbar space shows the posterior aspect of the fifth lumbar vertebra in contact with the posterosuperior aspect of the first sacral segment. The anterior variation is 1.45 cm. The fourth space shows the anterior aspect 0.95 cm. wider. The third and second spaces show equal variation of 0.80 cm. in their anterior aspects.

Figure 4 shows the dorsolumbar region in the erect posture, the tenth dorsal space being parallel, anterior and posterior aspects each being 0.55 cm. The eleventh dorsal space shows a variation of 0.02 cm., the twelfth 0.25 cm. and the first lumbar 0.35 cm., the anterior aspect in all cases being the wider.

Figure 5 shows the dorsolumbar region in flexion. The tenth dorsal space shows a variation of 0.05 cm., and the eleventh dorsal 0.20 cm., while the twelfth dorsal and first lumbar show no variation. The variation in each instance shows the pos-

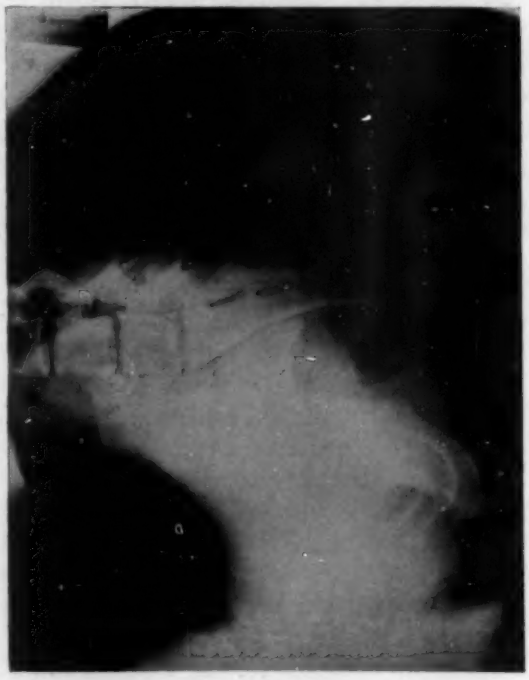


Fig. 2. Lumbosacral region. Extreme flexion (retouched).

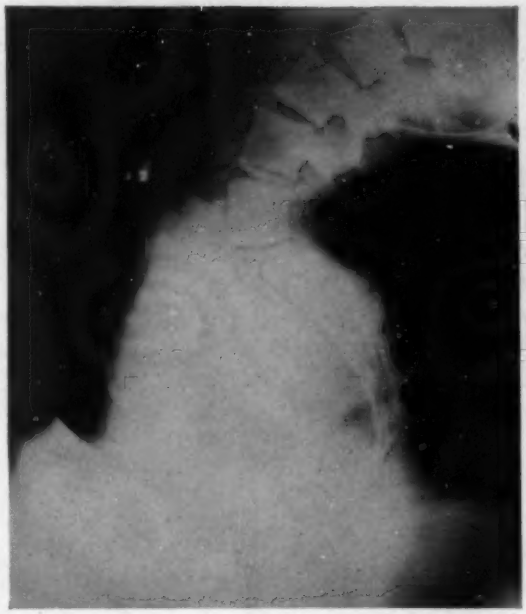


Fig. 3. Lumbosacral region. Extreme extension (retouched).

terior aspect wider. It is noted that the vertebral column and femora form an angle of less than 90 degrees.

Figure 6 shows the dorsolumbar region in extension. The tenth dorsal space shows a variation of 0.25 cm., the eleventh dorsal 0.50 cm., the twelfth dorsal 0.60 cm., and the first lumbar 0.85 cm. In all instances the anterior aspect is the wider.

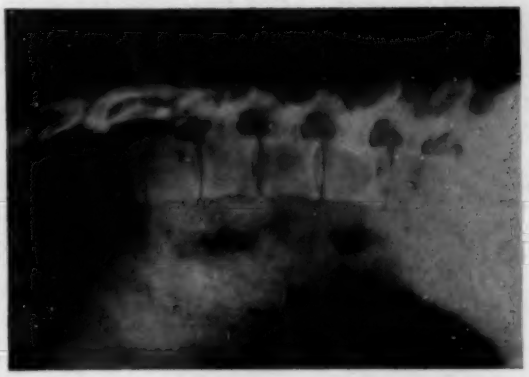


Fig. 5. Dorsolumbar region. Extreme flexion (retouched).

The range of motion in the spinous processes is of some interest. Those mensurable in this study in flexion and extension were the first, with a range of 1.85 cm., the second, 2.45 cm., third, 2.20 cm., and fourth, 2.20 cm.

The detailed measurements and averages are given in Table 1. In this table the range of motion in the lower dorsal and all the lumbar vertebrae equals the difference between the width of the intervertebral spaces anteriorly and posteriorly in extension and flexion. The average of the anterior aspects in 0.52 cm., and of the posterior aspects 0.32 cm.

The greatest range is in the anterior aspect of the second and fourth lumbar

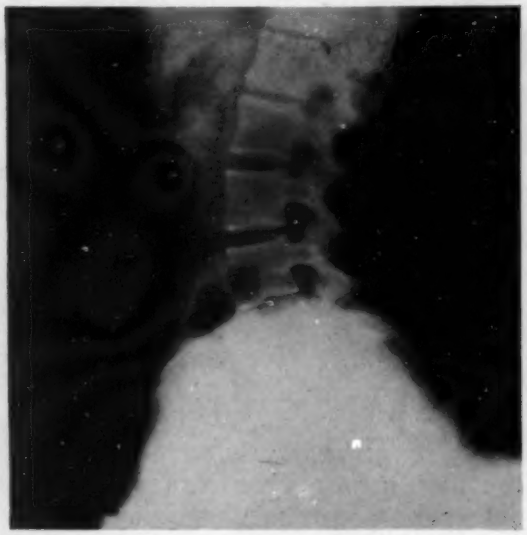


Fig. 4. Dorsolumbar region. Erect posture (retouched).

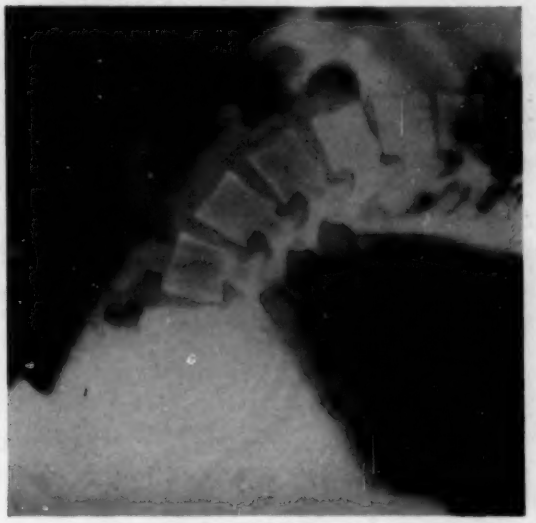


Fig. 6. Dorsolumbar region. Extreme extension (retouched).

Table I

Intervertebral Spaces	Erect Anterior Aspect	Erect Posterior Aspect	Flexion Anterior Aspect	Flexion Posterior Aspect	Extension Anterior Aspect	Extension Posterior Aspect	Range Anterior* Aspect	Range Posterior† Aspect	Spinous Process Flexion	Spinous Process Extension	Range Spinous Processes
	cm.	cm.	cm.	cm.	cm.	cm.	cm.	cm.	cm.	cm.	cm.
Tenth dorsal	0.55	0.55	0.45	0.50	0.70	0.45	0.25	0.05	1.90	**	**
Eleventh dorsal	0.60	0.58	0.40	0.60	0.95	0.40	0.55	0.20	2.10	**	**
Twelfth dorsal	0.75	0.50	0.60	0.60	1.00	0.40	0.40	0.20	0.20	**	**
First lumbar	0.95	0.60	0.70	0.70	1.25	0.40	0.55	0.30	2.35	0.50	1.85
Second lumbar	1.05	1.575	0.60	0.60	1.25	0.45	0.65	0.15	2.85	0.40	2.45
Third lumbar	1.025	0.65	0.70	0.85	1.25	0.45	0.55	0.40	2.60	0.40	2.20
Fourth lumbar	0.975	0.675	0.70	0.85	1.35	0.40	0.65	0.45	0.60	0.40	2.20
Fifth lumbar	1.10	0.80	0.95	0.80	1.45	0.00	0.50	0.80	**	0.20	**
Averages	0.875	0.616	0.637	0.687	1.15	0.368	0.52	0.3187	2.37	0.38	2.175

* Range: Anterior aspect in extension minus anterior aspect in flexion.
† Range: Posterior aspect in flexion minus posterior aspect in extension.
** Nonmensurable.
Anterior range of motion from tenth dorsal to fifth lumbar interspaces inclusive equals the anterior aspect in extension minus the anterior aspect in flexion. Average range 0.52 cm.
Posterior range of motion from tenth dorsal to fifth lumbar interspaces inclusive equals the posterior aspect in flexion minus the posterior aspect in extension. Average range 0.32 cm.
Average width of interspace of spinous processes in flexion 2.37 cm.
Average width of interspace of spinous processes in extension 0.38 cm.
These figures are not absolute, because the target-film distance is 100 cm. Distance of the subject in the erect posture, in flexion and in extension, has remained the same. The distortion is relatively constant.

spaces, these measuring 0.65 cm. each.

The figures given in the table are not absolute, the target-film distance being 100 cm. However, the distortion is constant, the distance factors in all instances being the same.

The only study in the literature comparable with the above is that of Dittmar, who undoubtedly did not consider the rôle of the hip joints when he stated that flexion is greater than extension in the lumbar spine. This statement is true only of motion of the torso as a whole on the thighs. It is definitely not true of intrinsic mobility of the lumbar spine. Moreover, extension is not greatest between the sacrum and fifth lumbar, the present study indicating equal range in the anterior aspects of the second and fourth lumbar spaces of 0.65 cm., the first lumbar space of 0.55 cm., and the lumbosacral of 0.50 cm. The intervertebral discs appear to alternately elevate and decline correspondingly anteriorly and posteriorly in flexion and extension, and vice versa.

This study confirms the work of Brailsford, and negates the statement of Dittmar in the particular instance specifically cited above. The statement of Lewis that flexion is freest in the lumbar region is disproved. The comment regarding mass movement of the torso on the thighs is applicable. The statement of Wiles and others concerning the displacement of vertebrae on adjacent fellows in flexion and extension was not confirmed by this study.

The spinal column is not a single hinge but a multiplicity of interacting hinges or, more precisely, limited universal joints, the nucleus pulposus acting somewhat in the manner of a ball-bearing. There is no single center of motion, except the mass motion of flexion centering in the hip joints. There are, however, numerous centers of motion, each with a limited range, the aggregate totaling the marked flexibility noted in special instances such as acrobats and contortionists, and also totaling in the normal individual quite sufficient mobility for the usual needs of the average person.

REFERENCES

1. Bishop. Researches into strength, flexibility, and curvatures of the vertebral column. *Lancet*, 1859, *2*, 470.
2. Blumenthal, M. Eine einfache Methode zur Darstellung und Messung von Körperbewegungen, insbesondere der Wirbelsäule. *Verhandl. d. deutsch. Gesellsch. f. orthop. Chir., Berl.*, xi. Kong., Stuttg., 1912, 107–127.
3. Bradford, E. H. Spinal flexibility. *Boston M. & S. J.*, 1922, *187*, 785–788.
4. Brailsford J. F. Radiology of Bones and Joints. J. & A. Churchill, Ltd., London, 1934.
5. Cyriax, E. F. An apparatus for estimating degree of rotation in the spinal column. *Brit. M. J.*, 1924, *2*, 958. Also, *Lancet*, 1924, *2*, 1024.
6. Davis, G. G. Applied Anatomy. Sixth edition. Revised by G. P. Muller. Philadelphia, 1924.
7. Dittmar, O. Die sagittal- und lateralflexorische Bewegung der menschlichen Lendenwirbelsäule im Röntgenbild. *Ztschr. f. d. ges. Anat. (Abt. 1)*, 1929, *92*, 644–667.
8. Engelhard, W. Die Haltung, Form und Beweglichkeit der Wirbelsäule in der sagittalen Ebene. *Ztschr. f. orthop. Chir.*, 1910, *27*, 1–16.
9. Guérin, J. Mémoire sur les mouvements de flexion et d'inclinaison de la colonne vertébrale. *Bull. Acad. de méd.*, 1876, *5*, 935; 1877, *6*, 155; 187.
10. Hall, H. J. Mobility of the normal spine in recumbency. *Tr. Am. Orthop. Ass.*, 1896, *9*, 190–194.
11. Heuer, F. Die Vor- und Rückwärtsbeugung der normalen Wirbelsäule unter besonderer Berücksichtigung der Aenderung, die der Bewegungsumfang in einzelnen Wirbelsäulen erleidet. *Ztschr. f. orthop. Chir.*, 1929, *52*, 374–387.
12. Heuer, F. Die physiologische und skoliotische Drehung der Wirbelsäule. *Ztschr. f. orthop. Chir.*, 1930, *52*, 513–533.
13. Hughes, A. W. Die Drehbewegungen der menschlichen Wirbelsäule und die sogenannten Musculi rotatores (Theile). *Arch. f. Anat. u. Entwcklngsgesch.*, 1892, 265–280.
14. Keene, C. W. Some experiments on mechanical rotation of the normal spine. *Am. J. Orthop. Surg.*, 1906–1907, *4*, 69–79.
15. Keller, H. Mobility of human spine. *Arch. Surg.*, 1924, *8*, 627–657.
16. Kellogg, L. C. Impossibility of moving vertebrae. *J. Am. M. Ass.*, 1923, *81*, 233.
17. Lewis, W. H., Editor. Gray's Anatomy of the Human Body. Philadelphia, 1936.
18. Löhr, C. Untersuchungen über die Bewegungen der Wirbelsäule nach vorn und hinten (nach einer neuen Methode am Lebenden). *München med. Wchnschr.*, 1890, *37*, 73; 97.
19. Lovett, R. W. Movements of normal spine in their relation to scoliosis. *J. Boston Soc. M. Sc.*, 1899–1900, *4*, 243.
20. Morris, H. The Anatomy of the Joints of Man. London, 1879.
21. Novogrodsky, M. Die Bewegungsmöglichkeit in der menschlichen Wirbelsäule. Dissertation, Bern, 1911.
22. Schmorl, G. Ueber die gesunde und kranke Wirbelsäule mit besonderer Berücksichtigung der Bandscheiben. *Genesek. bl. u. klin. en lab. v. d. prakt.*, 1932, *30*, 181–210.
23. Virchow, H. Ueber die sagittalflexorische Bewegung im Atlas-Epistropheusgelenk des Menschen. *Arch. f. Anat. u. Entwcklngsgesch.*, 1909, 294–299.
24. Virchow, H. Die sagittalflexorische Bewegung der menschlichen Halswirbelsäule. *Berl. klin. Wchnschr.*, 1919, *56*, 167.
25. Volkmann, A. W. Von der Drehbewegung des Körpers. *Arch. f. path. Anat. u. Physiol.*, 1872, *56*, 467–504.
26. Weber, E. H. Anatomisch-physiologische Untersuchung über einige Einrichtungen im Mechanismus der menschlichen Wirbelsäule. *Arch. f. Anat. u. Physiol.*, 1827, 240–271.
27. Wiles, P. Movements of lumbar vertebrae during flexion and extension. *Proc. Roy. Soc. Med.*, 1935, *28*, 647–651.
28. Wilson, P. D., and Cochrane, W. A. Fractures and Dislocations. Second edition. Philadelphia, 1928.
29. Winslow, J. B. Sur les mouvemens de la tête, du col, et du reste de l'épine du dos. *Hist. Acad. roy. d. sc. (de Paris)*, 1730, Amst., 1733, Mém., 492–508.

FRACTURES OF THE SACRUM

THEIR INCIDENCE IN FRACTURE OF THE PELVIS

By J. P. MEDELMAN, M.D.
SAINT PAUL, MINNESOTA

THIS review of cases was undertaken with the purpose of determining the incidence of fractures of the sacrum in association with fractures of other bones of the pelvic girdle. The subject is primarily

FIG. 1. Case 13. A fracture of the sacrum with displacement of the fragments. The fracture line follows the course through the sacrum which is most common whether or not the fragments are displaced.

one of academic interest because fractures of the sacrum are usually of secondary importance when they occur in conjunction with other fractures of the pelvis. They are not ordinarily displaced and are less apt to give rise to marked symptoms or to complications than the accompanying injuries. It is conceivable that such fractures may cause, either directly or indirectly, damage to the sacral plexus, rectum or other soft parts. Such an occurrence, however, is certainly exceptional. Because of the tendency of these fractures to remain in good position, they are frequently overlooked, both clinically and roentgenologically. Isolated fractures of the sacrum are apparently truly rare. However, due to the localization of symptoms, and the fact that they are frequently displaced to some extent, such fractures are apt to call attention to themselves.

A review of 125 cases of fractures of the pelvis by Noland and Conwell[1] in 1930 shows, in the whole group, only 3 cases of fracture of the sacrum with associated fractures of other bones of the pelvis. This is an incidence of only 2.4 per cent. In 1933 the same authors[2] presented a review of the original 125 cases, together with an additional group of 60 cases. In the second review they list 10 cases of the type under discussion. This means that 7 occurred in 60 fractured pelves which is an incidence of 11.7 per cent. On the other hand, Wakeley,[3] reporting from the clinical standpoint on 100 cases of pelvic fractures, presents 44 in which the sacrum was fractured in association with other pelvic bone fractures. These latter figures are exactly

FIG. 2. Case 33. A common type of sacral fracture.

the same as those obtained from a study of the group presented in this report.

I have reviewed the roentgenograms of the past 50 consecutive cases of pelvic fractures. They are tabulated in Table I. The anatomical distribution is shown more simply in Table II. This number of cases should be sufficiently large to give a good indication of the frequency of fractures of the sacrum. In the group of 50 cases, there are 22 in which the sacrum was fractured in

TABLE I

TABULATION OF CASES

No.	Body of Pubis	Superior Ramus	Inferior Ramus	Ala of Ilium	Acetab-ulum	Sacrum
1	R	R & L	R & L			L—Longitudinal
2			R			L—Longitudinal
3	L	R	R & L			
4		L			R	L—Longitudnal and horizontal
5		R & L	R & L			
6		R & L	R & L			L—Longitudinal
7	L	R & L	R & L			L—Longitudinal
8	R	L	L			
9		R	R			
10				R		
11		R	R			R—Longitudinal
12		R	R			
13	R	R & L	R & L			L—Longitudinal Markedly displaced
14		R	R & L			L—Longitudinal
15	L		L	L	L	L—Horizontal
16		L	L			
17	L	R & L	R & L			R—Longitudinal Slightly displaced
18		R & L	L			
19		R	R			R—Oblique
20		R & L	R			R—Oblique
21		R	R			
22		R & L	R			Sacroiliac separation
23				L		
24				L		
25		R	R & L			R—Oblique
26		R				
27	R		R			R—Longitudinal
28	R					
29						Transverse
30	L	L	L			L—Longitudinal
31					R	
32		R	R			
33	L					L—Longitudinal
34					R	
35			L			
36	L	R				R—Oblique
37	R					L—Longitudinal
38			L		L	L—Longitudinal
39	R	L	L			
40				R		
41	L	R	R			L—Oblique
42				R		
43			R		R	
44					L	
45			L		L	
46	R	R	R			
47		R & L	R & L			R—Longitudinal
48		R & L	R & L			R—Longitudinal
49		R	R			
50			R			

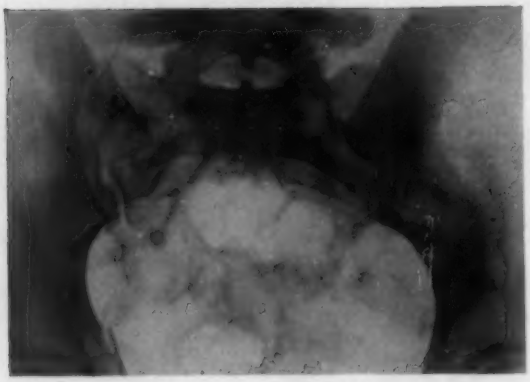


FIG. 3. Case 25. A sacral fracture similar to that shown in Figure 2 but with slightly more pronounced anatomical disturbance.

association with other fractures of the pelvis. This is an incidence of 44 per cent.

This incidence seems rather high considering the fact that such fractures receive so little attention and the statement in most textbooks that they are rare. However, there is nothing unusual about the type of the cases in the group. The injuries were sustained in a variety of ways and the injured individuals were not in any specialized industry or type of accident. It is to be noted that there is only one case in

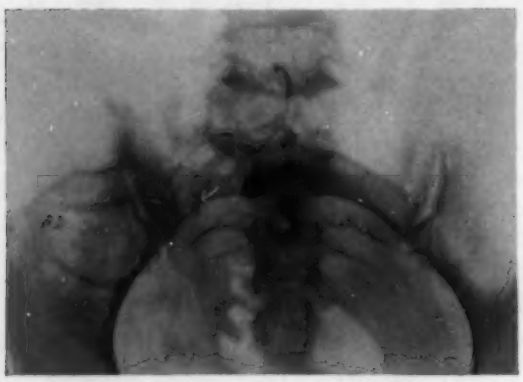


FIG. 4. Case 36. A fracture of the sacrum similar to those in the two preceding illustrations but with minimal roentgenological findings.

which the sacrum was the only fractured pelvic bone. This was a result of direct violence. In only one case was a definite sacroiliac separation suffered in the pres-

TABLE II

ANATOMICAL DISTRIBUTION OF PELVIC FRACTURES

	Right	Left	
Body of pubis	8	8	
Superior ramus	25	16	
Inferior ramus	26	20	
Ala of ilium	3	3	
Acetabulum	4	4	
Sacrum			
Associated with other fractures	9	13	
Isolated			1 (Transverse)
Sacroiliac separation	1		

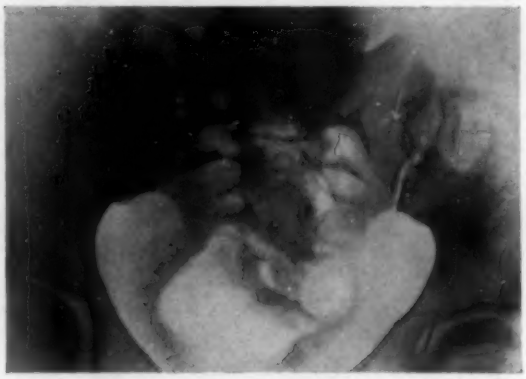


FIG. 5. Case 15. A horizontal fracture of the left side of the sacrum.

ence of fractures of the anterior portion of the pelvic girdle. This calls attention in a striking manner to the strength of the sacroiliac joint.

Only one side of the sacrum is involved in any case in this series except in the one case in which there is an isolated fracture of the sacrum. There seems to be no definite relationship between the side on which the sacral fracture occurs and the location of the fractures of the other bones of the pelvis. In a general way, those cases which show several fractures of the anterior half of the pelvis are more likely to show fractures of the sacrum. This is due to the fact that such cases undoubtedly were subjected to greater trauma and that consequently the shearing force exerted on the sacrum was greater.

The fractures are of three main types: longitudinal (the most frequent), oblique and horizontal. No case in this group shows a complete transverse fracture of the sacrum except the one in which the sacrum was the only fractured bone and in which direct violence was responsible for the injury. There is no appreciable degree of displacement of the fragments of the sacrum except in 2 of the cases. This rare occurrence of displacement is apparently due to the fact that the fractures usually are not complete. In the majority of cases, they can be detected in the roentgenograms extending through the bone only between adjacent anterior sacral foramina and then, in most cases, not completely through the longitudinal extent of the sacrum. In fact, a sharp angulation rather than the normal, smooth outline of the upper margins of one foramen or of adjacent foramina is the only sign that fracture is present in some of the cases. This can readily be accounted for by the relative thinness of the cortical bone of the sacrum and the fact that the cortex can be visualized in profile on anteroposterior views only at the upper margins of the foramina. These factors of minimal anatomical disturbance and the difficulty of visualizing such disturbance on roentgenograms are responsible for many fractures of the sacrum being overlooked.

CONCLUSIONS

1. Sacral fractures are not rare in association with fractures of other pelvic bones. They occur in 44 per cent of all cases in this group which is not a selected or unusual one.

2. Sacral fractures may easily be overlooked.

REFERENCES

1. Noland, L., and Conwell, H. E. Acute fractures of the pelvis. *J. Am. M. Ass.*, 1930, *94*, 174–178.
2. Noland, L., and Conwell, H. E. Fractures of the pelvis. *Surg., Gynec. & Obst.*, 1933, *56*, 522–525.
3. Wakeley, C. P. G. Fractures of the pelvis. *Brit. J. Surg.*, 1929, *17*, 22–29.

CONGENITAL PSEUDARTHROSIS OF THE TIBIA

By CLIFTON R. SCOTT, M.D.
NEW HAVEN, CONNECTICUT

CONGENITAL pseudarthrosis of the tibia and the prolonged dysfunction of the limb is a very perplexing problem to both the orthopedic surgeon and the roentgenologist and presents a complex group of clinical and roentgenological features concerning which little is known. Although a satisfactory classification of a disease is an essential procedure to a knowledge of it, the term osteitis fibrosa has been avoided for many reasons. In view of our present knowledge, it exists, both in its primary and secondary state, more as a condition or phase of normal bone reaction due to a multiplicity of causes and cannot be classified as a distinct clinical entity. We now know that the generalized process is distinctly a systemic one and is caused by or directly associated with hyperparathyroidism. The older writers recognized both a local and systemic process and believed them to be inflammatory in nature. As a name, theory or classification of disease is very hard to change because of history and usage, the title of this article might very properly read, congenital localized osteitis fibrosa cystica of the tibia, associated with pseudarthrosis.

The purpose of this communication is not to present any new theory or hypothesis concerning the etiological causation of this somewhat bizarre and rare localizing process but to present an extremely early and more or less typical case with the hope that it may stimulate some interest, if not in the causative factors, at least in the extremely essential early diagnosis and treatment of a comparative simple local process which causes such marked dysfunction, prolonged treatment and careful supervision.

Congenital deformity, bending and pseudarthrosis of the tibia has long been recognized as a distinct clinical entity, even though rare, and numerous cases have been reported, chiefly in the Italian and French literature. Because of the numerous and varied classification, it is extremely hard to give an accurate review of the cases reported. Wade[12] reports 10 cases, 3 seen before fracture occurred. Inglis[6] reports the pathology upon 3 of Wade's cases, 1, four days after birth. Henderson[5] reports 5 cases, successful operation being obtained in only 2 cases. Colonna[2] reports 3 cases, 1 of which eventually resulted in amputation.

The classification of the various cases is based almost entirely upon the clinical factors of varying deformities seen at different age periods. Gasne[4] gives the following classification: (1) well-marked anterior angulation, with no fracture at birth, (2) some slight deformity present at birth but developing fracture and pseudarthrosis following some slight trauma, (3) pseudarthrosis since birth, (4) normal at birth but later developing fracture and pseudarthrosis. Codivilla's[1] classification is much the same but he mentions very definitely the involvement of both the tibia and fibula associated with the presence of cyst-like zones with marked loss of bony substance.

In the past, normal bone tissue has been looked upon as a more or less static entity; however, in view of our present knowledge, we now know that even normal bone is constantly changing both in a macroscopic and microscopic appearance. Thus, bone may be greatly changed in size, shape, trabeculations and density at varying age periods of life under what may be termed normal living conditions. Consequently from the formation of the initial germ plasm to the death of the organism bone tissue is in a constant flux or change and many times it is very hard to differentiate normal physiological changes from those which might be termed pathological. Even the histogenesis of normal bone is very vague and unsettled. Two very definitely opposing views seem to predominate: (1) the so-called humeral theory presents the

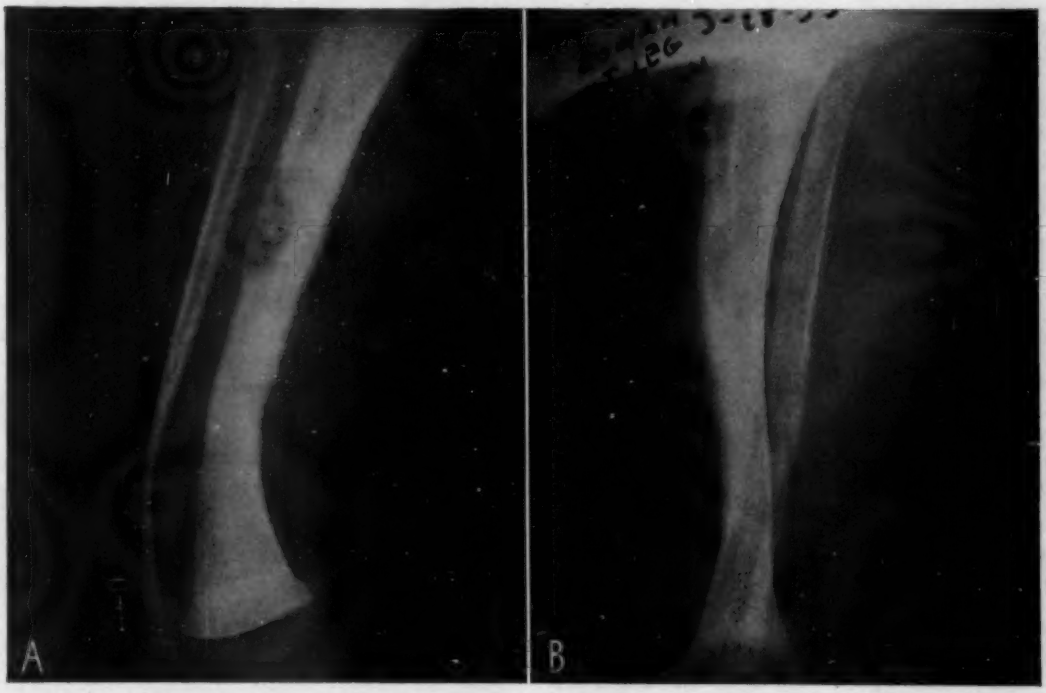


Fig. 1, *A* and *B*. Case 1. Fourteen days after birth. Note the large pseudocyst of the tibia and its relation to the point of entrance of the nutrient artery. Considerable outward bowing of the tibia and fibula, junction of the middle and lower thirds. Medullary space of both bones very narrow. At least some of this process must have been present before birth.

theory that bone is laid down and controlled to a large extent by the intercellular substance; (2) the so-called cellular theory of nuclear determinates. Both theories have their adherents and as our knowledge increases through research and experimental data we are led to the belief that there is much to be said in favor of each theory. Both closely overlap and are interrelated. Recently, Taylor,[11] in an experimental study of osteitis fibrosa, concludes that the calcifying power of the enzyme phosphatase in the osteoblast depends upon the quantity or concentration of the phosphatase in the cell itself and that decalcification is in reality a hindrance of recalcification because of the excessive removal of the enzyme due to hyperemia. In general, the formation of heterotopic bone and its reverse process necessitates the theory that connective tissue does and can form bone and that under certain conditions is interchangeable with endothelial cells, fibroblasts and bone cells. In other words, they are of the same general lineage, closely allied and under certain changes of environment may be interchangeable with each other. This opinion is the foundation or basis used in the classification of bone tumors proposed by Kolodny[8] and seems to be substantiated clinically.

Broadly speaking, the initial process is an intra-uterine metaplastic malacia and the essential feature is the formation of osteoid tissue limited entirely to the tibia and fibula. Absence of all fat with irregular bone foci which may coalesce into dense sclerotic masses or slowly disintegrate into pseudocyst-like areas may be present. These pseudocyst-like areas have a tendency to be cortical or subperiosteal in location, they are usually unilocular but may enlarge, coalesce or entirely disappear over varying periods of time. Any portion of the shaft may be involved at some time. These areas are supposed to be produced by ab-

FIG. 2. Case 1. Aged one and one-half months. Almost complete absence of the cyst of the tibia with a small, though well-defined cyst of the fibula, junction of the middle and lower thirds.

FIG. 3. Case 1. Aged one year. Fracture of the fibula and beginning cyst formation on the anterior surface of the tibia at the same level.

normal vascular changes, hemorrhage or the phagocytic action of newly formed giant cells. Apparently there is a very close connection between the formation of osteoid tissue, bone cyst and giant cell tumors as shown by various authors (Knaggs,[7] Platt,[10] Geschickter, Copeland, and Bloodgood[3]). Inglis[6] reports the pathology upon 3 of Wade's cases, 1 four days after birth, as showing typical osteoid tissue, poor vascularization and in certain sections frank

FIG. 4. Case 1. Aged one and one-half years. No new cysts but a further advance of the general process. Note the atrophy and pointing of the lower fragments of the fibula.

hemorrhage with pigment formation. In none of the cases reported was there seen small cell infiltration many times noted in the presence of infection. In the older age group there is well-marked avascularization but in none of the cases reported is there any direct evidence of any malformation of the nutrient artery to the bones although this feature has been presented as a causative factor or initiating influence in some cases. Knaggs[7] states that he is con-

vinced that there is an early bone destroying stage. This seems to be probable for osteitis fibrosa is essentially a reparative process. Codivilla[1] and Henderson[5] express the opinion that there may be a defective germ plasm and assume that there is some modification of the blood supply to the bone. That the vascular changes to any bone is a very important feature has been very clearly shown by Leriche and Policard.[9]

Fig. 5. Case 1. Aged two years and three months. New cyst high upon the lateral surface of the tibia. Marked narrowing of the medullary space.

The pathological fracture and the resulting dysfunction of the limb are what usually bring the patient to the physician, and in many cases no record or roentgenograms were made at birth or shortly thereafter. The fracture is almost always located at the junction of the middle and lower thirds of the tibia and, in the weight-bearing cases, is the result of the greatest static stress at a weakened point. Normally the blood supply to this portion of the tibia is very poor. There may or may not be pseu-

Fig. 6. Case 1. Aged two and one-half years. Fracture of the tibia at the junction of the middle and lower thirds.

Fig. 7. Case 1. Aged three years. Typical and well-marked pseudarthrosis of both tibia and fibula.

docysts present at this point. Body stance and posture, as in rickets, play their part. In early childhood the body stance and posture is essentially that of a gorilla: the center of gravity is high and well forward above the pelvis, the thigh is flexed upon the pelvis and the leg upon the thigh and the center of gravity and weight-bearing stress passes directly through the tibia at the junction of the middle and lower thirds. In an already weakened or pathological bone there is produced considerable anterior and lateral bowing, and one sees the direct application of Wolff's law in the extensive lamellar thickening, encroachment and narrowing of the medullary space and the avascularization which leads to fracture and non-union. These same factors explain the failure to obtain union by means of the massive bone graft in the early age group.

I have seen only 3 cases, 2 noted at a late age period and following a pseudarthrosis of some long duration. The other case seems to embrace all the clinical features of the disease and was fortunately seen at the early age period of fourteen days. The patient has been very carefully followed monthly with roentgenograms for a period of approximately four years and, although under very careful treatment and close supervision, a fracture occurred and a pseudarthrosis developed.

Case 1. B. C., female, was first seen at Grace Hospital fourteen days after birth. Pregnancy and delivery were normal as well as the initial examination of the infant. Approximately two weeks after birth some very slight inversion of the right foot was noted and roentgenograms were made at that time (Fig. 1). The patient was in excellent health and was gaining weight normally. Examination of the entire skeleton revealed the process to be limited entirely to the right tibia and fibula. Family history negative. The blood count was normal and the tuberculin and Wassermann reactions were negative. No calcium or phosphorus determinations were made as it was felt that the process was a purely local one and not systemic in nature. A diagnosis of congenital osteitis fibrosa cystica was made and the limb placed in a plaster cast. Many orthopedic consultations were obtained. The patient was followed very carefully and roentgenograms were made monthly. Casts were changed repeatedly and a metallic cuff or supportive splint was devised. In spite of all measures a fracture was predicted and occurred approximately two and one-half years after birth. The patient is now wearing a caliper splint and is approximately four years of age. Recognizing the condition, it was thought advisable that biopsy or bone grafting be postponed to a later age period.

The treatment recommended by all authors is delay and non-operative intervention in the early cases. General supportive measures with local physical therapy may be useful. The application of a plaster cast with pressure or the use of a metal cuff or splint may be useful in attempting to correct the early deformity. If seen during the weight-bearing period or following a definite pseudarthrosis, a caliper splint is most desirable in tending to correct the marked angular deformity which follows. Henderson[5] and Colonna[2] are firmly of the opinion that in all cases operative bone grafting before the age of six or eight is contraindicated and, if attempted previous to this period, will result in failure. Later, massive bone grafting usually produces union. Radiation therapy has been advised in some cases.

SUMMARY

Congenital osteitis fibrosa cystica and the subsequent pseudarthrosis is a somewhat rare and unusual condition. Few cases are reported or seen previous to the pathological fracture. The process is a purely local one and is present at birth as a cyst or osteoid tissue formation. Little is known concerning its initial formation. The pathological fracture is almost always at the junction of the middle and lower thirds of the bone and is probably caused by poor blood supply to this portion of the tibia. Operation is contraindicated prior to the age of six or eight years. It is only with the careful cooperation between the various clinicians that a correct diagnosis is made

and a good functioning leg achieved. Repeated roentgenograms are extremely useful in studying the varied cyclic changes of the osteoid tissue formation and definitely show the point at which fracture will subsequently occur.

REFERENCES

1. Codivilla, A. On the care of pseudarthrosis of the tibia by means of periosteal transplants. *Am. J. Orthop. Surg.*, 1906, *55*, 163–169.
2. Colonna, P. C. Congenital pseudarthrosis of the leg. *J. Am. M. Ass.*, 1934, *103*, 2012–2016.
3. Geschickter, C., Copeland, M. M., and Bloodgood, J. C. Osteitis fibrosa and giant cell tumor. *Arch. Surg.*, 1929, *19*, 169–271.
4. Gasne, E. Malformations symétriques des extrémités et particulièrement des pouces. *Rev. d'orthop.*, 1907, *8*, 298–302.
5. Henderson, M. S. Congenital pseudarthrosis of the tibia. *J. Bone & Joint Surg.*, 1928, *10*, 483–491.
6. Inglis, K. Pathology of congenital pseudarthrosis of the tibia. *J. Coll. Surgeons, Australasia*, 1928, *1*, 194–207.
7. Knaggs, R. L. The Inflammatory and Toxic Diseases of Bone. William Wood & Co., New York, 1926.
8. Kolodny, A. Bone Sarcoma: The Primary Malignant Tumors of Bone and the Giant Cell Tumor. Surgical Publishing Co., Chicago, 1927.
9. Leriche, R., and Policard, A. The Normal and Pathological Physiology of Bone. Translated by Sherwood Moore and J. Albert Key. C. V. Mosby Co., St. Louis, 1928.
10. Platt, H. Cysts of long bones of hand and foot. *Brit. J. Surg.*, 1930, *18*, 20–37.
11. Taylor, H. Osteitis fibrosa; an experimental study. *Brit. J. Surg.*, 1935, *22*, 561–588.
12. Wade, R. B. The so-called pseudarthrosis of the tibia. *J. Coll. Surgeons, Australasia*, 1928, *1*, 181–193.

PHYSIOLOGICAL EFFECTS OF RADIATION

III. THE LETHAL EFFECT OF ROENTGEN RADIATION PRODUCED BY VARIOUS KILOVOLTAGES (50 TO 1000 KV.) UPON THE BROWN-PEARCE RABBIT EPITHELIOMA*

By ROGER A. HARVEY,† RICHARD DRESSER, M.D.,‡ *and* STAFFORD L. WARREN, M.D.†
ROCHESTER, NEW YORK

THE present series of experiments were undertaken to extend the definition of the lethal *in vitro* massive roentgen-ray dosage for the Brown-Pearce rabbit epithelioma as a basis for an extensive study of the *in vivo* massive and fractional dosage systems, and to compare the effectiveness of radiation produced at 50, 90, 200, and 1,000 kv. upon this same tumor. The effect of roentgen rays of different wave lengths upon a given cell form or organism has long been a controversial point and has become more important with the introduction of apparatus which extends the available kilovoltage limits.

Much of the controversy regarding wave length effect has been caused by the variety of methods and objects used in tests. There is a rather marked lack of uniformity in the various techniques used, especially with regard to dosage measurements, elimination of back-scatter, etc. Skin is notorious for its individual physiological variations due both to type of skin (whether animal or human), the location and size of the area tested, and the amount of trauma to which it is unavoidably subjected—soilage, etc. *Drosophila* eggs and most animal tumors seem to be remarkably constant in their reactions in laboratories where their nature and peculiarities are well understood as a result of years of study.

From the enormous amount of experimental work[2] which has been done in the past, three theories have developed regarding the effectiveness of equal doses of the various wave lengths. The first is that within minor limits all ordinary wave lengths have the same effect. The second is that soft rays are more effective in small doses than hard rays in equal dosage (skin). The third is that there is a gradual increase in effectiveness with increase in wave length to a certain point and then a gradual recession in effectiveness.

Wood[12] studied the effectiveness of roentgen rays of two different wave lengths, 30–31 kv., and 100 kv. (0.2–0.17 Å) on rat and mouse tumors, and concluded that, in the investigated range, the intensity and not the quality of the roentgen rays was the important factor. Packard[4] showed that both homogeneous and heterogeneous beams of roentgen rays of the same intensity but of different wave length (0.22, 0.54, 0.68 Å) produced the same quantitative biological effects on *Drosophila* eggs.

Reisner and Neeff[5] stated that although equal doses of roentgen radiation having wave lengths from 0.14 to 0.26 Å were equal in reaction, longer waves were more effective.

Russ and Scott[6] claim that effectiveness increases with increasing wave lengths from a minimum to 0.45 Å.

The Brown-Pearce rabbit epithelioma was used in our experiments because of its stability since over 1,500 inoculations have demonstrated in our stock that it yielded a uniformly high percentage of takes (98 per cent), grew uniformly and rapidly, was highly malignant, and seemed relatively resistant to dosages of roentgen rays which were lethal to other animal tumors.[7]

* This investigation was carried out through a grant from the International Cancer Research Foundation.
† From the Division of Radiology, Department of Medicine, of the University of Rochester, School of Medicine and Dentistry, and the Strong Memorial Hospital, Rochester, N. Y.
‡ From the Collis P. Huntington Memorial Hospital, Boston, Mass.

METHOD

Method A: In view of our plan to study the various radiation dosage systems, it seemed feasible to investigate the minimal lethal dosage in massive form over the series of wave lengths at our disposal in order to utilize any advantages which might be derived from specific wave length effects if any were found.

In a previous paper of this series[7] there did not seem to be any definite difference in lethal effect when the tumor was irradiated (200 kv., heterogeneous beam, i.e., 0.5 mm. Cu, 1.0 mm. Al) *in situ*, *in vivo*, transplanted immediately, as an excised mass, or when cut up into fragments ready for inoculation. Therefore, in order that the tumor should be outside the body for the shortest possible time, it was irradiated *in situ* in the testicle and then transferred to other rabbits immediately. Since the lethal end-point with 200 kv. radiation was 6,000 r by this method, only the dosage range between 5,000 and 6,000 r was investigated.

50 kv. Radiation (0.274 Å Equivalent to 45 kv. (peak)). The roentgen-ray apparatus, which was constructed for experimental purposes, had four-valve tube rectification with oil immersed filament transformers in water-cooled tanks and a heavy duty transformer. An air-cooled Coolidge tube with tungsten target was used. The radiation factors were as follows: 50 kv., 5 ma., no filter, no cone, 15 cm. target-testes (mid-tumor) distance, and 60 r per minute. The dosage was determined with a Victoreen r-meter by placing the ionization chamber between the scrotum and penis at the level of a well-developed three weeks old (20 to 40 mm.) tumor mass in a stock animal. The testes at the level of the middle of the tumor masses were approximately 3 cm. apart. In this way, back-scatter due to the animal body and animal board was taken into consideration. Several dosage measurements were made before and after each application of roentgen rays. The r-meter was calibrated by the Victoreen Company for 50, 90, and 200 kv. levels.

An actively growing tumor mass, measuring at least 2 cm. in diameter and not more than three weeks of age, was left intact in both testes of the rabbits to be irradiated. The tumor-bearing testes were given a massive dose of 5,000 r after which one testis was aseptically removed and fragments of the tumor, all from the same level in the mass with respect to the target-tumor distance, were immediately inoculated into both testes of 7 rabbits. Immediately afterward, the remaining testis was given an additional 500 r for a total of 5,500 r, and its tumor likewise transplanted. Another rabbit-bearing tumor from the same generation, had one tumor-bearing testis removed and fragments of the tumor were inoculated into

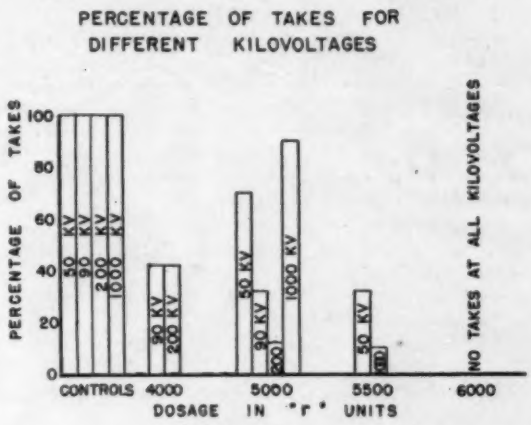


CHART 1

3 control animals. The remaining tumor-bearing testis was then given 6,000 r, and fragments of its tumor were transplanted into the testes of 7 rabbits.

In order to prove that failure of the irradiated tumor to grow in any rabbit was not due to some refractory state in the host but to the irradiation of the tumor cells, every rabbit in which growth of the irradiated tumor failed to occur was later inoculated with untreated tumor fragments. Before any of these rabbits were reinoculated, 80 to 120 days were allowed to elapse to allow for extremely long latent periods.

Results with 50 kv. Radiation. The *percentage of takes* (Chart 1 and Table 1) or growth resulting from inoculation of the irradiated tumor gives a clear-cut indication of the lethal effects of irradiation. All 6 control inoculations grew. The general average for the groups showed 98 per cent takes over the last three years. Tumor treated with 5,000 r (14 implants) showed 71 per cent takes; that treated with 5,500 r showed 35.7 per cent takes; that treated with 6,000 r (8 implants) showed no takes. This shows the relatively slight effect of irradiation with 5,000 r, the more marked effect of 5,500 r, and the completely lethal effect of 6,000 r.

TABLE I

EFFECT OF IRRADIATION UPON GROWTH FACTORS

Kilovoltages	CONTROLS					4000 r					5000 r					5500 r					6000 r	
	Number of Implants	Percentage of Takes	Average Latent Period	Average Growth Period	Average Length of Life	Number of Implants	Percentage of Takes	Average Latent Period	Average Growth Period	Average Length of Life	Number of Implants	Percentage of Takes	Average Latent Period	Average Growth Period	Average Length of Life	Number of Implants	Percentage of Takes	Average Latent Period	Average Growth Period	Average Length of Life	Number of Implants	Percentage of Takes
50	6	100	6.5	33.3	40						14	71	25	36.5	63	14	35.7	42	44.5	90	8	0
90	3	100	7.0	39.3	45.6	8	43.7	34	43.7	73.7	9	33	30.3	53.4	79.2						8	0
200*	26	100	6.9	45.0	52.0	18	27.7	27.4	54	77	22	13.6	20.3	48	68.3						30	0
1000†	3	100	8.3	35.3	43.6						10	90	23.8	48.3	68.2	10	10	70	67	137	10	0
Av. Total	38	100	7.2	38.1	45.3	26	35.7	28.7	44.5	75.3	55	63.1	24.8	47.5	71.9	24	22.8	56	52.6	113	56	0

* From Sahler and Warren.[7]
† By R. Dresser.

The lethal end-point is thus demonstrated to be between 5,500 and 6,000 r.

The *latent period* (Chart II and Table I), that is, the length of time between the date of inoculation of the tumor and growth of the tumor to a palpable size, more readily demonstrates the effect of irradiation upon the tumor cells. Controls showed a latent period of six to seven days which was consistent for this tumor generation. Tumor treated with 5,000 r showed a latent period varying from twenty-one to thirty-five days with an average of twenty-five days. Tumor irradiated with 5,500 r showed a latent period varying from thirty-five to forty-nine days. This is a clear example of the fact that within the critical dosage levels, increased doses of roentgen rays change the latent period markedly not only by reducing the number of viable cells but also by interfering with their reproductive characteristics.

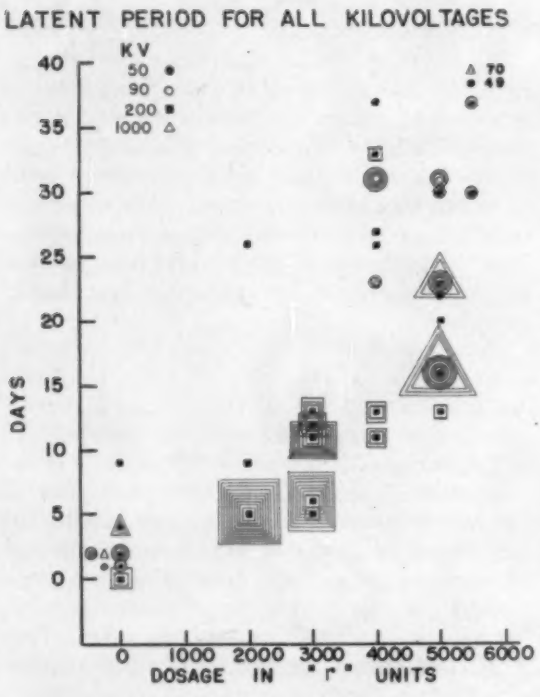


CHART II

Charting the *growth characteristics* shows only a slight effect of 5,000 r upon the growth rate of the tumor once the growth has become re-established after treatment, for four of the ten curves lie well within the normal range (Chart III). The average growth (lethal) of the control tumors was 11.6 mm. per week (14.5 to 8.5 mm. being the extremes), while the average length increase was 8.1 mm. per week (extremes 12.5 to 4.6 mm.) in the 5,000 r group, and 5.1 mm. per week (10.0 to 1.2 mm. for the extremes) in the 5,500 r group (Table II).

The *length of life* of the rabbit also shows a progressive increase as the dosage of roentgens is increased (Table I). Animals inoculated with tumor treated with 5,000 r varied in their life span from 42 to 120+ days. In those in which

TABLE II

AVERAGE GROWTH OF LONG DIAMETER DURING ACTIVELY GROWING PERIOD

	CONTROLS	No.	Av.	4000 r	No.	Av.	5000 r	No.	Av.	5500 r	No.	Av.
50 kv.	14.5 12.2 12.2 12.0 10.0 8.5	6	11.6				12.5 6.0 12.5 5.5 9.3 4.6 8.7 7.7 7.4 7.2	10	8.1	10.0 9.0 3.4 2.0 1.2	5	5.1
90 kv.	19.2 14.8 13.3	3	15.9	17.5 15.5 15.0 13.0 13.0 10.6 3.3	7	12.5	12.0 11.2 10.0 9.3 9.3 9.0	6	10.1			
200 kv.	14.0 10.0 11.0 13.0 11.5 11.0 12.5 6.0 12.3 13.5 5.5 18.6 9.3 9.6 9.3 12.0 12.5 13.0	18	11.3	8.6 5.0 8.0 2.0 7.6 7.0 6.6 6.5 5.6 5.0	10	6.0	9.5 9. 8.6	3	9.0			
1000 kv.	15.6 13.6 11.4 10.8 9.2	5	12.1				18.0 6.8 11.7 6.3 9.5 8.7 8.0 8.0 7.3	9	9.4	7.2	1	7.2
Gen Av.		19	12.75		16	9.9		28	9.15		6	6.15

death was due to cachexia from tumor during the course of the experiment, this varied from forty-three to eighty-one days with an average length of life of sixty-three days. In the case of tumor treated with 5,500 r, only 2 animals died from tumor cachexia during the course of the experiment at the end of eighty-seven and ninety-three days respectively. Five animals in these two groups outlived the course of the experiment (120+ days) and were omitted here as elsewhere from the length of life analysis because of the static character of the growth. Animals inoculated with tumor treated with 6,000 r all outlived the duration of the experiment. When the average lengths of life of sixty-three days for the 5,000 r group, and ninety days for the 5,500 r group are compared with the control group of forty days, the marked effect of irradiation on increase in the length of life is noted.

The period of palpable growth or the measurable actively growing period (Table 1) also shows the effect of radiation for the different levels (excluding static tumors). The controls showed palpable growth periods varying from thirty-two to thirty-five days with an average

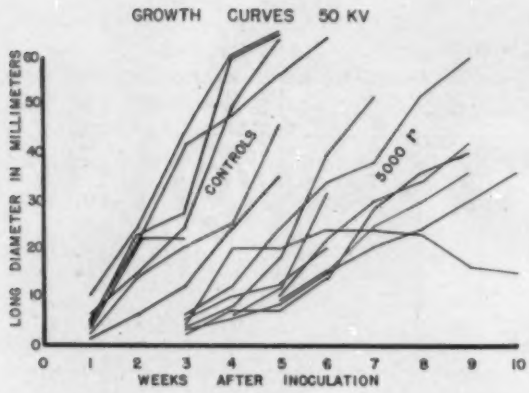


CHART III

of 33⅓ days. The group treated with 5,000 r showed a palpable growth period varying from twenty-two to fifty-three days not including the 2 cases which survived the 120-day period of the experiment with static tumor. The average for this group is 36.5 days, and is so near the control average of 33⅓ days that it is well within the range of experimental variation and seems to indicate that when doses of roentgen rays below the critical or lethal level for the tumor are given, very little slowing of the active growth period occurs (Chart IV). However, the shortened growth average per week (8.1 mm.) and the static status of the tumor in the 2 cases which survived the duration of the experiment does suggest a sustained effect from the irradiation in some irradiated tumor fragments. In the group treated with 5,500 r, one tumor fragment had a palpable growth period of forty-four days, another forty-five days, and 2 showed static tumor at the end of the experiment. Damage to the tumor is also reflected by the slower average weekly increase in length, i.e., 5.1 mm. (Table II). Although the number of cases is small, there is at least an indication here that, as the dosage approaches the lethal level, the palpable growth period is increased.

90 kv. Radiation (0.144 Å Equivalent to 85 kv. (peak)). The method used for investigating the radiation produced at this kilovoltage was identical with that of the previous section, except that the radiation factors were 90 kv., 1 mm. Al filter, 5 ma., 50 r per minute, no cone, and 20 cm. target-mid-tumor distance. Tumor masses from the same generation were irradiated as described for 50 kv. and fragments inoculated in a single testis. Eight single testis inoculations were used for the 4,000 r dosage, 9 testis inoculations for 5,000 r, and 8 for 6,000 r. Three unirradiated fragments were inoculated for controls. Refractoriness to the tumor was ruled out by reinoculation in cases where the tumor transplant did not appear after 120 days.

All of the irradiated groups of tumor showed a smaller percentage of takes than the controls. Tumor treated with 4,000 r showed takes in 43.6 per cent; at 5,000 r, 33 per cent; and at 6,000 r there were no takes at all (Table I). Therefore, there is a marked decrease in the percentage of takes at 4,000 r, and a slightly greater decrease at 5,000 r. The lethal endpoint is demonstrated to be between 5,000 r and 6,000 r.

The *latent period* (Table I and Chart II) between the time of inoculation and the appearance of the palpable tumor mass demonstrates evidence of damage from the irradiation. Controls showed an appearance time of from six to eight days after inoculation. The irradiated cases in which growth took place vary in their appearance time from twenty-one to forty-two days with an average of thirty-four days for the 4,000 r group, and 30.3 days for the 5,000 r group (Table I and Chart II). Most of the irradiated tumor transplants showed a *slower growth rate* for the first few weeks after the tumor became palpable than did the corres-

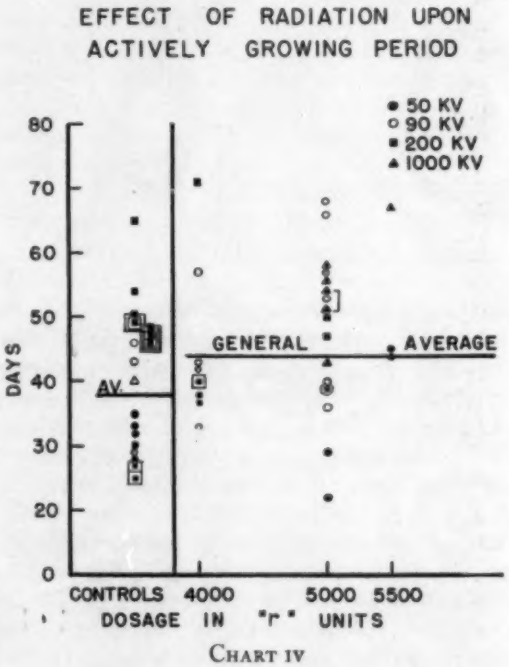


CHART IV

ponding controls (Table II and Chart V). The average increase in length for the controls was 15.9 mm. per week (extremes 19.2 to 13.8 mm. per week) while the average for the 4,000 r group was 12.5 mm. per week (extremes 17.5 to 10.6 mm. per week with one case of 3.3 mm. per week) and the 5,000 r group averaged 10.1 mm. per week (extremes 12 to 9.0 mm. per week). However, after the initial slow rate of development the growth rate seemed to approach that of the controls. This initial delay in growth is reflected in the length of the actively growing period.

The *length of life* of all of the animals inoculated with irradiated tumor was markedly longer than that of the controls (forty-five days). The average length of life of the group irradiated with 4,000 r (seventy-three days) was not greatly different from that of the 5,000 r group (seventy-nine days)—Table I.

The changes in the measurable *actively growing period* (Chart III) brought about by the irradiation resembled those noted in the 50 kv. experiment. The 4,000 r and 5,000 r groups showed the development of some static tumors without metastases developing. The averages are similar and show definite increase (43.7 days for the 4,000 r; 53.4 days for the 5,000 r, in contrast to 39 days for the controls). Even though the static tumor-bearing animals are omitted from this analysis, these periods are increased and indicate a definite tumor damage, although greater numbers might increase the spread in the shorter intervals.

200 kv. Radiation (0.63 Å Equivalent to 196 kv. (peak)). The results from the use of this kilovoltage are taken from a previous paper[7] because, although the tumor generation is that of the year previous to the start of these ex-

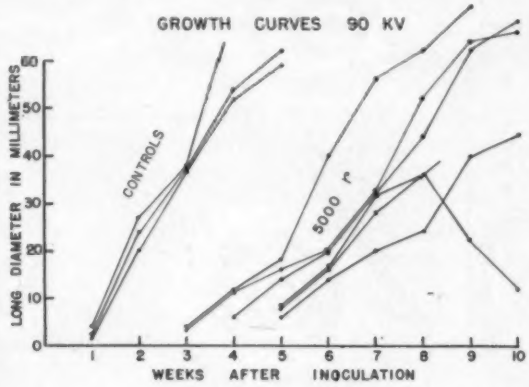


Chart V

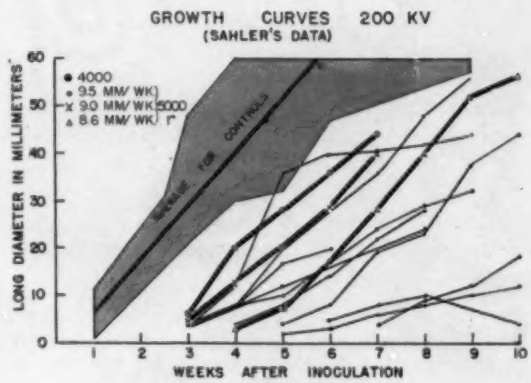


Chart VI

periments, the method employed is essentially the same.

The radiation factors were 200 kv., 25 ma., filter 0.5 mm. Cu plus 1 mm. Al, 25 cm. circular cone, 28 cm. target-mid-tumor distance, and 165 r per minute. Dosage was determined with a Victoreen r-meter by placing the ionization chamber between the penis and scrotum at the mid-tumor level. The tumor was irradiated *in situ* and fragments from the same level with respect to the beam were transplanted immediately.

The *number of takes* dropped from 100 per cent for the controls to 83 per cent for 2,000 r and 3,000 r; 27 per cent for 4,000 r; 13 per cent for 5,000 r; and none at 6,000 r (Chart I).

The *latent periods* (Table I and Chart II) increased from an average of seven days for the controls to twenty-three days for 4,000 r, and twenty days for the 5,000 r group.

The growth rate per week, i.e., length (Table II and Chart VI) averaged 11.4 mm. per week for the controls (extremes 14.0 to 5.5 mm. per week); for 4,000 r, 7.3 mm. per week (extremes 8.6 to 5.0 mm. per week); and for 5000 r, 9.0 mm. per week (extremes 9.5 to 8.6 mm. per week). In general, the growth rate for the irradiated fragments was delayed at first. The growth rate curves (Chart VIII) are nearly parallel to those of the controls. This is reflected in the relatively slight increase in the length of the *actively growing period* (forty-five days for the controls; fifty-four days for 4,000 r; and forty-eight days for the 5,000 r groups—Table I and Chart IV).

1,000 kv. Radiation. Since the beginning of our investigations of irradiation effects on the Brown-Pearce rabbit epithelioma, supervoltage apparatus for the production of roentgen rays

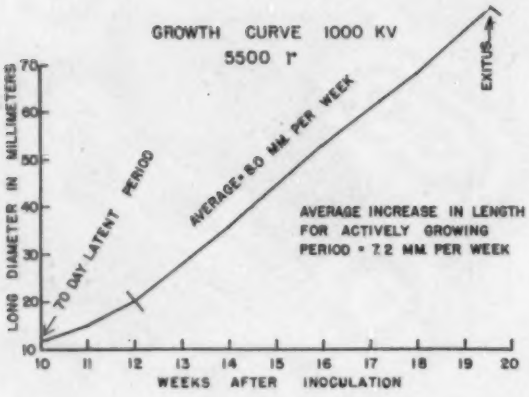

GROWTH CURVE 1000 KV
5500 r
LONG DIAMETER IN MILLIMETERS
70 DAY LATENT PERIOD
AVERAGE 8.0 MM. PER WEEK
AVERAGE INCREASE IN LENGTH FOR ACTIVELY GROWING PERIOD = 7.2 MM. PER WEEK
EXITUS
WEEKS AFTER INOCULATION

CHART VII

has been put into operation. In order to include the higher range of kilovoltage, arrangements were made to have animals, bearing the tumor, irradiated with roentgen rays produced at 1,000,000 volts. The results of this experiment furnish a good check upon the results obtained with radiation produced at 50, 90, and 200 kv., because the dosage measurements and irradiation were done by different workers and in a different laboratory from the preceding experiments. The tumor generation used was comparable to that used for the 50 kv. experiments.

Method. Three rabbits bearing tumor masses 2 cm. in length in each testis were shipped to the Collis P. Huntington Memorial Hospital in Boston for irradiation. One animal received 5,000 r over the testicular region, another received 5,500 r, and a third received 6,000 r. After treatment, the animals were immediately returned by express. Fragments of the irradiated tumor were inoculated into both testes of 5 rabbits for each dosage level, employing the same technique as in all the previous experiments. Three controls from the same stock inoculated from the same tumor generation were also transplanted in the same way. In the investigation with lower kilovoltage irradiation, the tumor was inoculated immediately after treatment, but in this case there was a delay of fifteen hours between the time of irradiation and inoculation due to transit. The irradiation factors employed were as follows: 1,000,000 volts, constant potential, 35 cm. target-tumor distance, 4 mm. of lead filter, plus 3 mm. brass plus the lead and copper in the anode, making an equivalent of 6 mm. of lead total filtration. The generator was operated at 1 ma. which gave an output of 240 r per minute. The measurements were made with a thimble chamber (Victoreen condenser Type) calibrated for this radiation, with the animal board in place, that is with back-scatter taken into account. No measurements are available for the shortest wave length for this beam.

Results with 1,000 kv. Radiation. The results of this experiment are divided into the usual five groups for analysis. Since the results are of considerable interest, they are described in detail even though they are in no way different from those obtained with the other voltages.

5,000 r: Rabbit 1564 first showed palpable tumor on the twenty-first day in both testes. Growth was rapid and death occurred on the forty-ninth with numerous metastases. Rabbit 1565 also showed palpable tumor on the twenty-first day in both testes. Growth was rapid and exitus occurred on the sixty-fourth day with several metastases present. Rabbit 1566 also showed palpable tumor on the twenty-first day in both testes. Growth was rapid and death occurred on the seventy-seventh day with numerous metastases present. Rabbit 1567 showed palpable growth in the left testis on the twenty-first day and in the right testis on the twenty-eighth day. Growth was rapid and exitus occurred on the seventy-second day with numerous metastases. Rabbit 1568 first showed palpable growth in the left testis on the twenty-first day. Growth was slow and exitus took place on the seventy-ninth day with numerous metastases. There was no growth in the left testis.

Thus there were 10 inoculations with 9 growths of tumor, or 90 per cent takes from the tumor receiving 5,000 r (Chart 1).

5,500 r: Rabbit 1569 showed palpable growth

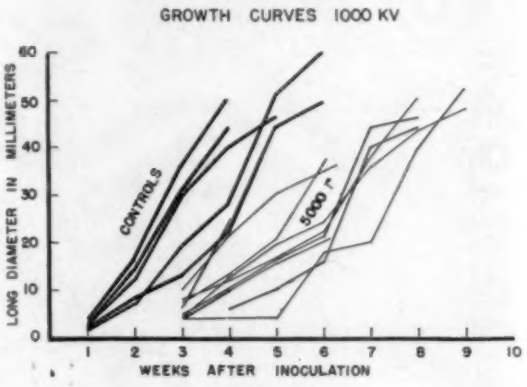


CHART VIII

in the left testis on the seventieth day. Growth was slow at first but later nearly paralleled the rapidity of growth of the slower controls (7.2 mm. per week—Chart VII). Death occurred on the 137th day from cachexia with extension of the tumor up the spermatic cord, a few metastases in the lungs, under the skin of the neck and in the left eye, and many fair-sized nodules in the retroperitoneal lymph nodes and kidneys. The tumor at the site of inoculation measured 8.5×2.6×2.8 cm. and was firm and regular in contour. Approximately 40 per cent of it was translucent (actively growing tumor tissue). This represents a fairly good growth rate for the sixty-seven days of growth from the time of appearance (seventy days latent period).

Rabbits 1570, 1571, 1572, and 1573 all failed to show growth of the tumor treated with 5,500 r. Thus there were 10 inoculations with growth of only one tumor, or 10 per cent takes.

6,000 r: Rabbits 1574, 1575, 1576, 1577, and 1578 failed to show growth of the inoculation of tumor treated with 6,000 r. Thus there were 10 inoculations of tumor fragments with no growth of the tumor (Chart I and Tables I and II).

After inoculation with unirradiated tumor, growth of the tumor took place in all of the rabbits which failed to develop tumors from irradiated fragments (rabbits 1570 through 1578) indicating the absence of refractory states.

In brief, the tumor treated with 5,000 r showed 90 per cent of takes; that treated with 5,500 r showed 10 per cent takes; and that treated with 6,000 r showed no takes (Chart I), while all three control fragments took and grew in the usual manner.

The controls had a *latent period* (Table I and Chart II) of seven to nine days while the fragments of the tumor irradiated with 5,000 r showed a latent period varying from twenty-one to twenty-eight days (averaging 23.8 days). The one fragment which grew after irradiation with 5,500 r appeared after a latent period of seventy days.

The *length of life* of the animals is also dependent upon the dosage of roentgen rays given to the tumor. The animals inoculated with tumor treated with 5,000 r showed a life span varying from forty-nine to seventy-nine days with an average of 68.2 days. None of the animals inoculated with tumor irradiated with 5,500 r or 6,000 r died during the course of the experiment (120 days+)—Table I.

The growth rate of the tumor nodules, once the tumor became palpable and growth well established in the new host after irradiation, showed very little change (Chart VIII) from the control series. The controls grew in length at the rate of 12.1 mm. per week (extremes being 15.6 to 9.2 mm. per week) while the 5,000 r group averaged 7.6 mm. per week (extremes being 14 to 5 mm.)—Table II. The *actively growing period* was increased only thirteen days—from thirty-five days for the controls to forty-eight days for the 5,000 r group. That for the one survivor of 5,500 r, which grew rather slowly at first, grew fairly rapidly after the first two weeks of palpable growth (the first two weeks at 4 mm. per week, and then at 8.1 mm. per week with a general average of 7.2 mm. per week for the sixty-seven days until death from cachexia occurred)—Table II.

Thus the results of this experiment are comparable with the results obtained in the previous experiments performed at 50, 90, and 200 kv.

DISCUSSION

The Brown-Pearce rabbit epithelioma was used for this investigation because more than three years' work with it in this laboratory had demonstrated that it would give a consistently high percentage of takes, would metastasize widely, had a fairly constant latent period and life-span, and seemed consistently resistant to roentgen irradiation. A constant source of susceptible rabbits was also a factor for our confidence in the regularity of the response of the tumor. Several months after completing this series of experiments (May, 1937) we were forced to resort to heterogeneous sources for our rabbits and soon began to notice a change in the behavior of the tumor. The percentage of takes was normal, but the latent period, the rate of growth and the length of life were markedly prolonged and the number of metastases were minimal. Experimental work was stopped at this point and our endeavors aimed toward reviving the virulence of the tumor. Rabbits from various sources were inoculated, and a strain of susceptible

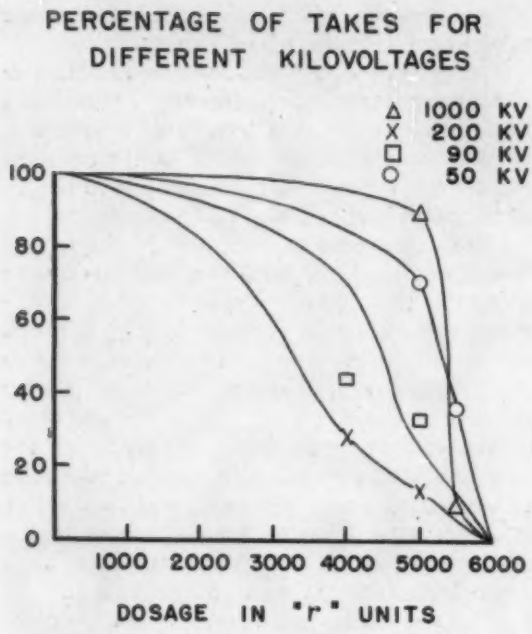


CHART IX

rabbits has now revived the tumor to its former status (February, 1938). Other workers who have had long experience with this tumor have undoubtedly encountered a similar situation[1]. This change in the status of the stock made it impossible to amplify the experiments further and fill in the gap in the 5,500 r groups of experiments with 90 and 200 kv. radiation. However, the results seem to be sufficiently complete to allow considerable analysis to be made and certain fairly clear-cut conclusions may be drawn without further experiment.

An analysis of the *percentage of takes* is probably the most important. The controls showed the same high percentage of takes which was consistent in our stock over the whole time of the various experiments. The percentage of takes of irradiated implants varied considerably at different dosage levels with the various kilovoltages used, but more than 5,500 r was necessary to prevent takes in all experiments (from interpolation of Chart IX for 90 and 200 kv.). The variations between experiments at the 5,000 r level are not difficult to explain. The 200 kv. experiment was performed a year earlier and the tumor generation therefore cannot be too closely compared with the other experiments. There was a fifteen hour interval in the 1,000 kv. experiment between irradiation and inoculation, a factor not present in any of the other experiments. The relatively few inoculations at each level, however, are probably the greatest factors because one failure to take would alter the results from 8 to 14 per cent, depending upon the number of inoculations. The number of animals used (Table 1), on the other hand, was quite large for an experiment as unwieldy as this. A study of the percentage of takes indicates no marked superiority of the radiation produced at 1,000 kv. over that at 50 kv., or at any other voltage, under these experimental conditions. The same quantity of radiation at each kilovoltage is required for complete failure of takes, specifically more than 5,000 r and less than 6,000 r.

Increases in increment of dosage of 250 r between 5,000 and 6,000 r would be impracticable because 250 r represents only 4.16 per cent of the total dosage. Such small increases in dosage would yield little definite information because errors in measurement (±5 per cent) and variations in individual biological reactions would more than offset a 5 per cent difference in dosage. A dosage of 500 r represents 10 per cent of 5,000 r and 8.3 per cent of 6,000 r, and is a reasonable increment of change in experiments of this type. An analysis of the rate of change in destructive effect of the radiation produced at the various kilovoltages is of some interest (Chart X) although within the refinements of these experiments there is certainly no demonstration of gross difference in destructiveness among the wave lengths used. Chart IX shows the number of takes plotted in percentages, the curves being fitted as well as possible to the experimental points by means of a spline. Although we have just stated that increments of dosage of 500 r are the smallest practical divisions for the biological aspect of the experiment, these

curves are drawn empirically on a large scale and increments of change (in percentage) for units of 250 r were used to give a smooth curve. The 200 kv. curve (Chart x) falls to the left and the 50 and 1,000 kv. curves are superimposed to the right of the 90 kv. curve. Since the experiments were done on different tumor generations, this fact would seem to have more influence upon the position of the peaks of the curves than any peculiar wave length effect. The 200 kv. experiments were done in October, 1935; the 90 kv. experiments in November, 1936; and the 50 and 1,000 kv. experiments in March, 1937. The tumor generation has a very definite relationship to the growth characteristics of the tumor surviving irradiation since the lengths of the actively growing periods of the controls (Table 1) show a corresponding shift (the controls for 200 kv. had an actively growing period of 45.0 days, the 90 kv. group of 39.3 days, while the 50 and 1,000 kv. groups had periods of 33.3 and 35.3 days, respectively). The growth curves of these experiments show similar groupings (Charts III, V–VIII) although they are more erratic.

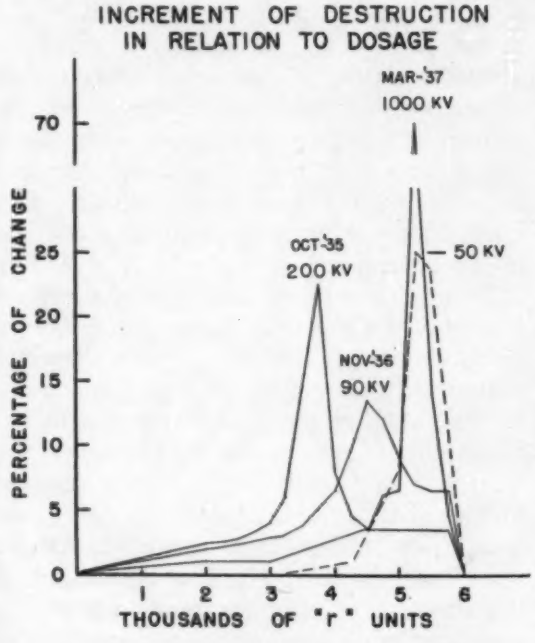


Chart X

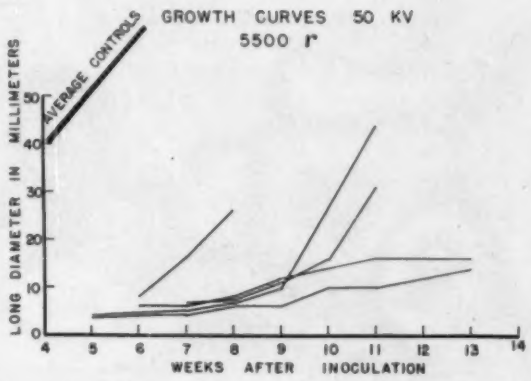


Chart XI

We defined the latent period as the lapse of time between the inoculation of the tumor fragment and the appearance of a palpable tumor nodule. This lapse of time obviously does not represent a period of inactivity on the part of the tumor. It merely represents the interval required for sufficient cells to proceed through enough generations to produce an aggregate of palpable size. In the unirradiated controls, there are numerous cells from the very start which serve as sources for ensuing generations. In the case of irradiated tumor, the number of viable cells or cells capable of reproduction is reduced by the application of roentgen rays so that from the time of inoculation there are fewer potential cells which can serve as a basis for development of a nodule of comparable size with the control. From a study of the latent periods produced by the various dosages and wave length levels, we must conclude that with increasing dosages the latent period is increased without respect to the investigated range of wave length. Increase in the average latent period is probably a better index than the percentage of takes of the damage (complete or partial) produced by the irradiation (Chart II).

The growth characteristics of the tumor fragment transplanted after irradiation have been analyzed by plotting the length of the tumor nodule at weekly intervals. In a large number of controls it was found that when length measurements were plot-

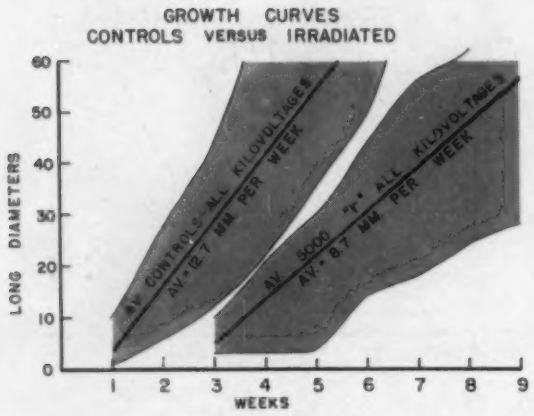


CHART XII

ted they have a curve almost identical to the mean volume curve of the same tumor nodule.[7] This nodule is restricted by anatomical barriers in its lateral growth in the testis after a time, but is not hampered appreciably in the length of growth up to death of the animal from cachexia. Since this measurement is much simpler to use and is probably as accurate as any volume estimate that could be made under the conditions of measurement, we have used it throughout. The control inoculations of a tumor fragment generally show a slightly steeper and more uniform slope in the growth curves than the irradiated inoculations. All of the irradiated inoculations appear later, and tend to have a slower growth rate for a few days after appearance and then proceed to grow at the same rate or only slightly slower than that of the controls. The initial lag in growth after appearance is more marked as the the lethal end-point of dosage is approached as shown in the 50 kv. chart for the 5,500 r level (Chart XI). Once the tumor nodules treated with 4,000 and 5,000 r have passed through the latent period and the phase of slow growth, the rate becomes more like that of the controls. After 5,500 r at 50 kv., the growth rate is much slower than the controls. The one case which grew after 5,500 r at 1,000 kv. showed a latent period of seventy days and then grew at a rate nearly parallel to the controls. This is a confirmation of similar observations by Hill, Morton and Witherbee.[3] There does not seem to be any apparent difference in growth rate whether 50, 90, 200, or 1,000 kv. are used for 5,000 r (Chart IV). There does seem to be some sustained diminution in growth rate after 5,500 r, especially in the 50 kv. experiment. This is well demonstrated in Chart XII where the control growth curves (averaging 12.7 mm. per week) would be overlapped by the 5,000 r growth curves (all kilovoltages) to a very large extent if the latent period is subtracted (if the curves are shifted to the left 3 units of time). Except for the presence of a few slow-growing tumors in the 5,000 r group (lowering the right hand edge of the pattern) the chief reason for a lower average of 8.7 mm. per week is the period of slow growth during the first few weeks of palpable growth (Charts III, V–VIII). Table II gives all the average growth rates per week for the various experiments and the total averages for the dosage levels.

It is surprising to find that 5,000 r (83.3 per cent of the lethal dose) will affect the growth of an irradiated transplant so slightly that its growth (if it survived) is only partially affected and then only at first, so that once growth is well established it may return to the activity of the controls. A study of the frequency and number of metastases occurring from surviving tumor transplants irradiated with 5,000 and 5,500 r at all kilovoltages also does not indicate any reduction in the frequency and number of metastases below that occurring in the controls.

It would appear that the chances of killing all of the cells, leaving some alive but permanently damaged, others alive but temporarily damaged, and perhaps some entirely undamaged is based upon a probability dosage-cell number relationship (the "number of chance hits") rather than a matter of accurate dosage. Since the *in situ* irradiation-transplantation method causes some mechanical damage to the cells and the subsequent struggle to survive in the new host must cause some decrease in the number of takes, we should expect a higher

lethal dosage range for strictly individual *in vivo* irradiation experiments. This has been borne out by preliminary experiments of this type to be reported in a subsequent paper of this series.

CONCLUSIONS

1. The percentage of takes of irradiated tumor material decreases with increasing dosage of roentgen rays within the critical range so that more than 5,000 r and less than 6,000 r are required for complete lethal effect. No marked effect of wave length was noted.

2. The latent period is prolonged with increase in dosage of roentgen rays, but a wave length effect is not appreciable.

3. Growth curves of irradiated tumor show a slower rate of increase than the unirradiated controls, but in a few cases, growth rate of irradiated tumor may resume the characteristic control rate after a lapse of several weeks.

4. The actively growing period of irradiated tumor material is not appreciably greater than that of control tumor once growth has become palpable in both cases.

We wish to express our appreciation to Francis W. Bishop for his assistance and advice in conducting the irradiation experiments, and to Clarence A. Du Pont for checking the wave length measurements in the 50, 100, and 200 kv. experiments.

REFERENCES

1. Casey, A. E. Reaction of standard breeds of rabbits to Brown-Pearce tumor. *Am. J. Cancer*, 1937, *31*, 446–456.
2. Duggar, B. M., Editor. Biological Effects of Radiation. McGraw-Hill Book Co., New York, 1936 Chap. xiv, p. 473.
3. Hill, E., Morton, J. J., and Witherbee, W. D. Studies on x-ray effects; direct action of x-rays on transplantable cancers of mice. *J. Exper. Med.*, 1919, *29*, 89–96.
4. Packard, C. Biological effectiveness of high-voltage and low-voltage x-rays. *Am. J. Cancer*, 1932, *16*, 1257–1274.
5. Reisner, A., and Neeff, T. C. Hauttoleranzdosis und Strahlenqualität. *Strahlentherapie*, 1929, *34*, 313–339.
6. Russ, S., and Scott, G. M. Differential action of x-rays and gamma rays upon some living tissues. *Brit. J. Radiol.*, 1929, *2*, 301–306.
7. Sahler, O. de P., and Warren, S. L. Physiological effects of radiation. II. The *in vitro* lethal single massive roentgen-ray dosage (at 200 kv.) for the Brown-Pearce rabbit epithelioma. Am. J. Roentgenol. & Rad. Therapy, June, 1939, *41*, 954–961.
8. Schrek, R. Method for counting viable cells in normal and in malignant cell suspensions. *Am. J. Cancer*, 1936, *28*, 389–392.
9. Sugiura, K. Reaction of transplantable mouse sarcoma No. 180 to radiations of different wave lengths (200 kv. roentgen rays and gamma rays). Am. J. Roentgenol. & Rad. Therapy, 1934, *31*, 614–627.
10. Sugiura, K. Study of back-scattered radiation and depth dosage using transplantable animal tumor as indicator. *Radiology*, 1936, *26*, 76–85.
11. Wood, F. C. Further studies in the effectiveness of different wave lengths of radiation. *Radiology*, 1925, *5*, 199–205.
12. Wood, F. C. Effect on tumors of radiation of different wave lengths. Am. J. Roentgenol. & Rad. Therapy, 1924, *12*, 474–482.

THE RELATION BETWEEN TIN AND COPPER FILTRATION WHEN USING A THICK-WALLED ROENTGEN TUBE*

By M. C. REINHARD *and* H. L. GOLTZ
BUFFALO, NEW YORK

THORAEUS has shown that the use of tin filters to produce a beam of roentgen rays of the same quality as one produced by a copper filter results in a gain in intensity of 25 per cent. This work was carried out with thin-walled glass roentgen tubes. Subsequently, Thoraeus pointed out that the superiority of tin filtration over copper filtration is somewhat less in the cases of thick-walled tubes. In corroboration of this last statement we have found that, when using a thick walled G.E. XPT tube within an XPT tube shield, the superiority of a tin filter over a qualitatively equivalent copper filter (half-value layer = 1.72 mm. copper) is approximately 13 per cent.

Since some radiologists have attempted to harden a beam of 200 kv. roentgen rays by means of extreme filtration beyond the above mentioned quality, it was decided to investigate the relative merits of copper and tin as filters in this range, using as a source of radiation a thick-walled tube.

All measurements were made with a Victoreen condenser r meter and thimble ionization chamber, suspended in air. The roentgen tube was operated at 200 kv. (peak) and 25 ma.

Starting with a filter of 0.5 mm. copper, intensity measurements were made with copper filters up to 9.0 mm. The results obtained from these measurements were plotted in a curve (Fig. 1). From this curve the half-value layers in millimeters of copper were obtained for successively increasing thicknesses of copper filtration. This relationship is shown in Figure 2.

Similar to the procedure followed in obtaining the data for Figure 1, measurements were made using various increasing thicknesses of tin as a filter. These data

* From the State Institute for the Study of Malignant Disease, Buffalo, N.Y., Burton T. Simpson, M.D., Director.

TABLE I

COMPARISON OF TIN AND COPPER FILTRATION OF AN XPT TUBE IN AN XPT TUBE SHIELD OPERATED AT 200,000 VOLTS AND 25 MA.

Cu Filtration		Half-Value Layer	Tin Filtration		Gain	λ eff.*
mm. Cu 1 mm. Al	r/min. (air)	mm. Cu	mm. Tin 0.25 Cu 1 mm. Al	r/min. (air)	Per Cent	
1.0	26.7	1.23	0.21	28.0	4.9	0.142
1.5	19.3	1.47	0.33	21.5	11.4	0.130
2.0	15.0	1.72	0.47	16.8	12.0	0.120
2.5	11.9	1.89	0.62	13.5	13.4	0.114
3.0	9.6	2.19	0.78	11.0	14.6	0.105
3.5	7.9	2.35	0.92	9.4	19.0	0.100
4.0	6.7	2.47	1.07	8.2	22.4	0.097
4.5	5.8	2.55	1.20	7.2	24.1	0.095
5.0	5.0	2.61	1.30	6.6	32.0	0.094
5.5	4.4	2.66	1.39	6.1	38.7	0.093

All thicknesses of metal were determined by a micrometer taking an average of ten readings for each thickness.
* λ eff. taken from Grebe-Nitzge tables for the corresponding half-value layers in millimeters of copper.

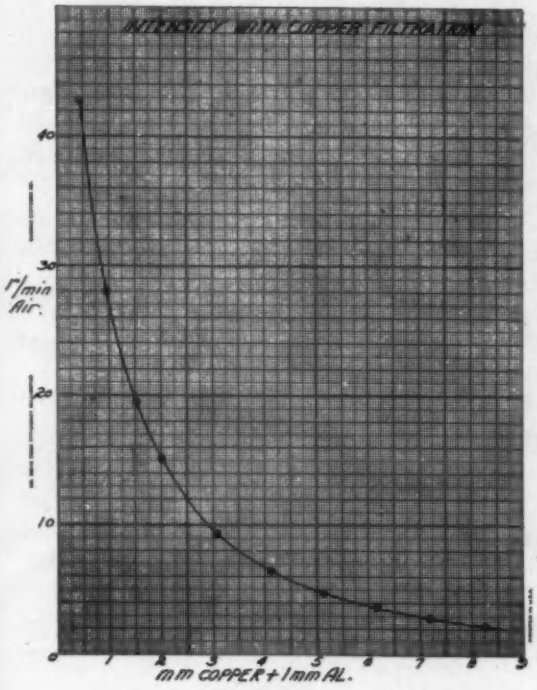


FIG. 1

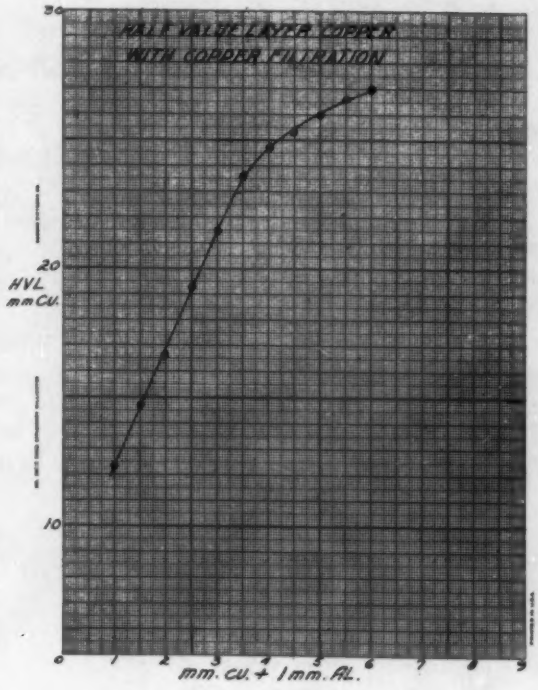


FIG. 2

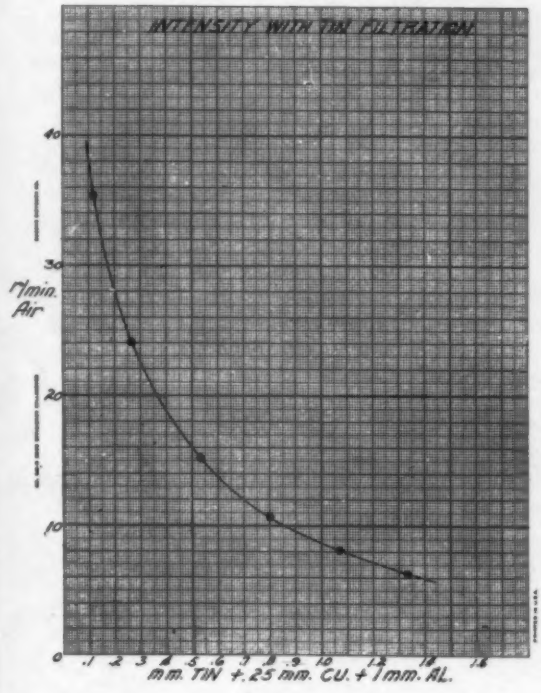


FIG. 3

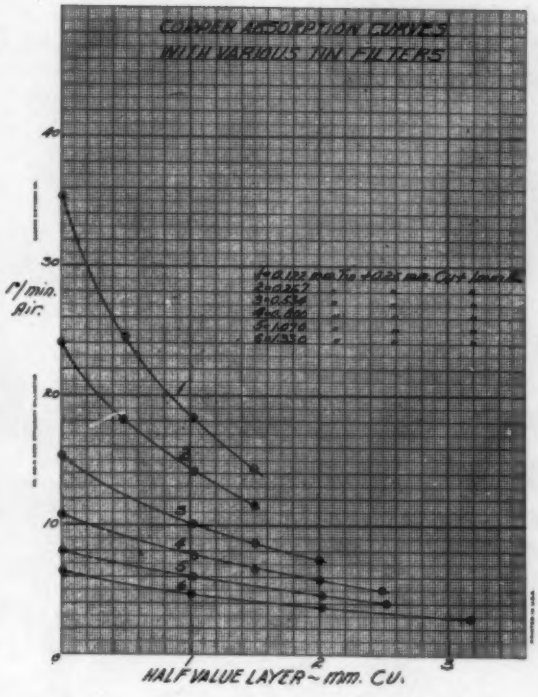


FIG. 4

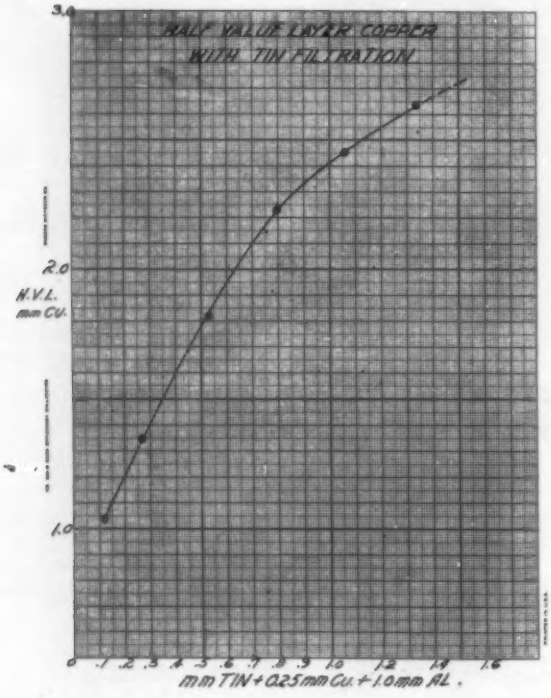


FIG. 5

are expressed graphically in Figure 3. In addition to the tin filters, a filter of 0.25 mm. copper and 1 mm. aluminum was always present.

In order to obtain the half-value layer in millimeters of copper for the various tin filters, several thicknesses of tin, arbitrarily chosen, were used and the half-value layer of copper determined for each thickness of tin. These absorption curves are shown in Figure 4.

Following the procedure used in obtaining from Figure 1 the data expressed in Figure 2, the half-value layers in copper were obtained for increasing thicknesses of tin from Figure 4 and are shown in Figure 5.

From Figures 1 to 5 the data tabulated in Table 1 were obtained. This table is self-explanatory and shows that, for qualitatively equivalent filters, the superiority of tin over copper gradually increases from 4.9 per cent for a beam having a half-value layer of 1.23 mm. copper, to 38.5 per cent for a beam having a half-value layer of 2.66 mm. copper.

NOTE: This paper is not intended as a recommendation that the extreme filtration mentioned herein is a satisfactory method of producing harder radiation. In fact, a glance at Figure 2 shows that above a filter of 3.4 mm. copper, the curve falls off very rapidly.

Vol. 42, No. 1

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

Editor: Lawrence Reynolds, M.D. *Assistant Editor:* Ruth Bigelow, B.S.

Editorial Board: A. C. Christie, M.D. E. H. Skinner, M.D. Lauriston S. Taylor
Advisory Board for Pathology: James Ewing, M.D. Eugene L. Opie, M.D.
Collaborating Editors: The Officers and Committee Members of the Societies of which this Journal is the official organ, whose names appear on this page, are considered collaborating editors of this Journal.
Foreign Collaborators: A. Béclère, M.D., Paris, Gösta Forssell, M.D., Stockholm, G. F. Haenisch, M.D., Hamburg, R. Ledoux-Lebard, M.D., Paris.

Publisher: Charles C Thomas, 220 East Monroe St., Springfield, Illinois.

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application: Editorial office, 110 Professional Building, Detroit, Mich. Office of publication 220 E. Monroe St., Springfield, Ill. Information of interest to all readers will be found on page iv.

Officers and Standing Committees

THE AMERICAN ROENTGEN RAY SOCIETY

President: J. W. Pierson, Baltimore, Md.; *President-Elect:* E. L. Jenkinson, Chicago, Ill.; *1st Vice-President:* W. McD. Doughty, Cincinnati, Ohio; *2d Vice-President:* C. G. Dyke, New York City; *Secretary:* C. B. Peirce, Royal Victoria Hospital, Montreal, P.Q., Canada; *Treasurer:* J. B. Edwards, 144 Woodridge Place, Leonia, N. J.; *Historian:* Percy Brown, 475 Commonwealth Ave., Boston, Mass.

Executive Council: J. W. Pierson, E. L. Jenkinson, W. McD. Doughty, C. G. Dyke, C. B. Peirce, J. B. Edwards, Lawrence Reynolds, H. D. Kerr, S. W. Donaldson, H. E. Ruggles, C. A. Waters, B. R. Kirklin, R. C. Beeler, R. S. Bromer, U. V. Portmann, Chairman, Cleveland Clinic, Cleveland, Ohio.

Committee on Hospital Relations: J. D. Osmond, Cleveland, Ohio, F. F. Borzell, Philadelphia, Pa., John Sproull, Haverhill, Mass., D. S. Childs, Syracuse, N. Y., L. C. Kinney, Chairman, San Diego, Calif.

Committee on Safety and Standards: R. R. Newell, San Francisco, Calif., Otto Glasser, Cleveland, Ohio, Douglas Quick, New York City, E. C. Ernst, St. Louis, Mo., Lauriston S. Taylor, Chairman, Washington, D. C.

Publication Committee: E. P. Pendergrass, Philadelphia, Pa., M. C. Sosman, Boston, Mass., H. E. Ruggles, Chairman, San Francisco, Calif.

Finance Committee: H. J. Walton, Baltimore, Md., Robert Drane, Savannah, Ga., H. D. Kerr, Chairman, Iowa City, Ia.

Committee on Scientific Exhibits: V. W. Archer, University, Va., P. A. Bishop, Philadelphia, Pa., S. W. Donaldson, Chairman, 326 N. Ingalls St., Ann Arbor, Mich.

Representatives on The American Board of Radiology: B. R. Kirklin, Rochester, Minn., U. V. Portmann, Cleveland, Ohio, LeRoy Sante, St. Louis, Mo.

Member, National Research Council: Ross Golden, New York City.

Editor: Lawrence Reynolds, 110 Professional Building, Detroit, Mich.

Assistant Editor: Ruth Bigelow, 110 Professional Building, Detroit, Mich.

Editorial Board: A. C. Christie, E. H. Skinner, Lauriston S. Taylor.

Advisory Board for Pathology: James Ewing, Eugene L. Opie.

Fortieth Annual Meeting: Palmer House, Chicago, Ill., Sept. 19–22, 1939.

THE AMERICAN RADIUM SOCIETY

President: Lawrence A. Pomeroy, Cleveland, Ohio; *President-Elect:* Frederick W. O'Brien, Boston, Mass.; *1st Vice-President:* Hayes E. Martin, New York City; *2nd Vice-President:* Eugene T. Leddy, Rochester, Minn.; *Secretary:* William E. Costolow, 1407 S. Hope St., Los Angeles, Calif.; *Treasurer:* Charles L. Martin, Baylor Hospital, Dallas, Texas.

Executive Committee: Zoe A. Johnston, Chairman, Pittsburgh, Pa., E. H. Skinner, Kansas City, Mo., W. P. Healy, New York City.

Program Committee: F. W. O'Brien, Chairman, Boston, Mass., Leland Cowan, Salt Lake City, Utah, W. S. MacComb, New York City, A. N. Arneson, St. Louis, Mo.

Publication Committee: E. H. Skinner Chairman, Kansas City, Mo., H. G. F. Edwards, Shreveport, La., A. Strauss, Cleveland, Ohio.

Research and Standardization Committee: Wilhelm Stenstrom, Chairman, Minneapolis, Minn., E. P. Pendergrass, Philadelphia, Pa., Otto Glasser, Cleveland, Ohio.

Education and Publicity Committee: W. P. Healy, Chairman, New York City W. E. Howes, Brooklyn, N. Y., G. Allen Robinson, New York City.

Advisory Committee on Roentgen-ray and Radium Protection: Edith H. Quimby, Chairman, New York City, E. C. Ernst, St. Louis, Mo., H. J. Ullmann, Santa Barbara, Calif.

Janeway Lecture Committee: Zoe A. Johnston, Chairman, Pittsburgh, Pa.

Committee on Arrangements: Douglas Quick, Chairman, New York City, Maurice Lenz, New York City, Hayes E. Martin, New York City.

Representatives on American Board of Radiology: Douglas Quick, New York City, R. H. Stevens, Detroit, Mich., B. P. Widmann, Philadelphia, Pa.

Delegates to the Sixth International Congress of Radiology: Albert Soiland, Los Angeles, Calif., Delegate, E. C. Ernst, St. Louis, Mo., Alternate Delegate.

Twenty-fifth Annual Meeting: New York City, 1940.

EDITORIALS

THE ANNUAL MEETING

THE Fortieth Annual Meeting of the American Roentgen Ray Society will be held at the Palmer House, Chicago, Illinois, on September 19, 20, 21 and 22, 1939, under the Presidency of Dr. John W. Pierson. Elsewhere in this issue will be found the preliminary program which has been arranged by the President-elect, Dr. E. L. Jenkinson, and the Program Committee. The program covers a wide range of subjects dealing with lesions of the gastrointestinal tract, roentgen studies of the neck and of the heart and aorta, and a very large part is given over to diseases of the respiratory tract as well as to diseases of the genitourinary tract, and of the skeletal system. Ample provision has been made for a comprehensive discussion of the physics of radiation and the results of roentgen and radium therapy.

The Caldwell Lecture, always an outstanding feature of the annual meeting, is to be given by Dr. James T. Case, who has chosen as his subject "Roentgenology of Pancreatic Disease." This should be a most stimulating discussion of a somewhat neglected field of roentgenology. Dr. Case, who is Professor of Radiology in the Medical School, Northwestern University, brings to this lecture a wealth of experience and observation. Dr. Jenkinson is peculiarly fortunate in his selection of the Caldwell lecturer as it was Dr. Case himself who many years ago suggested the propriety and the importance of establishing a lectureship in memory of the late Eugene Caldwell.

Dr. Jenkinson and his committee have made a departure in the arrangement of the program in that all of the scientific papers are to be given in the morning, leaving the afternoon free for the Instruction Courses, a detailed program of which was given in the June issue of the JOURNAL. Those who have not made application for these Instruction Courses are advised to do so immediately as it may be seen from a study of the outline of these courses that they are well planned and that the subjects which are to be discussed are of fundamental importance to all radiologists. Dr. B. R. Kirklin, who arranged the Instruction Courses, has made a special effort to secure men of outstanding ability to make them interesting as well as instructive.

The usual social affairs incident to the meeting have been well thought out. On Monday will be the Golf Tournament at the Flossmoor Country Club, at which time the players will compete for the Willis F. Manges Trophy. Those who are interested should make application immediately to the Chairman of the local Golf Committee, Dr. M. J. Hubeny, Cook County Hospital, Chicago. On Tuesday evening will be held the annual banquet at which time will occur the presentation of the gold medal of the Society and following the banquet there will be dancing.

The Scientific Exhibit which is being arranged by Dr. S. W. Donaldson and his committee promises to be an excellent one. The exhibit hall at the Palmer House offers an unusual opportunity for a proper display of such an exhibit. The Commercial Exhibit which is always an important feature of the annual meeting will be of special interest not only in the increase in the number but in the quality of the exhibits.

Those who anticipate attending the meeting should make their reservations at once, since Chicago with its central location will stimulate increased attendance.

Photograph by Hornbaker, Scranton

BYRON HUBBARD JACKSON
1873–1939

DR. BYRON H. JACKSON of Scranton, Pennsylvania, a pioneer practitioner of radiology, whose recent death is mourned by the myriad of his scientific colleagues, entered upon the work of his specialty at almost the exact middle of that romantic decade of discovery, research and development which immediately followed the revelation of roentgen rays in 1895. Therefore for him, as for many medical men of contemporaneous background, the turn of the century marked the inception of a new professional career—a career which was embraced with avidity by some as a permanent and radical departure; by others, tried and later relinquished, in

whole or in part, when it seemed destined to displace clinical or laboratory activities already established. History records that Byron Jackson, with that singularly lucid foresight of which the pioneer is so frequently possessed, became promptly an unalloyed devotee to the "new science" as soon as he was convinced as to the beneficial effects of radiation in the determination or treatment of disease.

Doctor Jackson, a son of Jabez and Samantha (Mathers) Jackson, was born in Kingston, Pennsylvania on September 22, 1873. Developing at the public schools into an earnest and steadfast student with an early predilection for the career of a physician, he was graduated from the University of Maryland with its Medical Class of 1898. He began his professional work as a general practitioner in the rural Pennsylvania districts of Harvey's Lake and Mayfield, but removed to Scranton about the year 1900 to enter into the practice of radiology in association with the late Dr. John Price. Imbued, as has been said, with the spirit of the moment so easily remembered today by all who once shared its dominating fascination, he developed more and more definitely a leaning toward radiology as his life work—work that has now reached its culmination so suddenly and so sadly, yet so honorably.

There is little doubt that Byron Jackson encountered the discouragements and the external resistances so common to the experience of the enthusiast who endeavors to establish a new and highly specialized branch of practical investigation in any community, particularly a small one. Difficult indeed his first forward steps, without benefit of established precedent, with but weak support from recent acquaintances among new colleagues and, above all, by no means exempt from an early and widespread incredulous cynicism toward his specialty and all its works. Jackson, however, was one of those whose persistent determination, defying such inhibitions, soon received proper recognition. His colleagues in other fields of medical work became increasingly impressed by the advantage of his painstaking cooperation in the solution of their clinical problems, and his reputation as a conservative and dependable radiologist widened as his experience increased. Far beyond the confines of his own community the influence of his work was felt by organized groups of radiologists, with which his affiliation was welcomed. In these connections he took the highest satisfaction, which, by reason of his warm personality and his high executive ability, was shared universally by others. In consequence, his contributions to the furtherance of organization activities were of the greatest value. In 1908, he was elected to membership in the American Roentgen Ray Society, but eight years after its founding. In 1920, he was one of a group of early members of the Radiological Society of North America instrumental in effecting its reorganization during that year. Doctor Jackson gave to this newer Society the preponderance of his energy and loyal devotion, which very properly led to his election as its President for 1933.

This year of 1933 was for him one of great executive activity in many ways. It marked his able participation, as Chairman of a Committee on History and Education of the First American Congress of Radiology, in the preparation of a volume entitled "The Science of Radiology," which appeared in connection with the autumn meeting of this newly organized Congress. Also during the course of the same year, Doctor Jackson served with distinction as President of the Radiological Society in fulfilment of his election at its meeting of 1932. Ever jealous of the good name of American Radiology and with high ideals concerning her status and its permanent clarification, it was again in 1933 that he added the influence of this high office to the establishment of the American Board of Radiology.

Previous to this, Doctor Jackson had been created a Fellow of the American College of Radiology and the American College of Physicians. In 1932, he was made

a Corresponding Honorary Member of the Royal Society of Medicine (Section of Radiology) of London. He was also a Member of the American Medical Association, the Pennsylvania Radiological Society and of the Philadelphia Roentgen Ray Society. Of the Lackawanna County Medical Society he was the President for the usual term of one year; he was its Librarian for several years.

Jackson's daily occupation as a radiologist within the community wherein he practised was both varied and arduous, but pursued by him with the genuine pleasure that complete devotion engenders; consequently his efforts were abundantly fruitful in positive results. By the benevolent acts of his professional life he proved that the essential humaneness of Medicine can be as clearly revealed in necessarily restricted specialism as in the broadest application of general practice. For twenty years he served faithfully as Radiologist-in-Chief at the State Hospital and also for a long period of time in the same capacity at the Hahnemann Hospital, at Scranton. So great did his activity become that, aside from his extensive private practice, he was in charge, at the time of his death, of the radiological services at the Moses Taylor, the West Side, and St. Mary's Hospitals, in the same city. These duties never appeared to bear heavily upon his capable shoulders, so eager seemed he in his willingness to serve and in his glad alacrity of manner toward the fulfilment of his professional tasks, however onerous. He possessed an imperishable philosophy of cheerfulness which was transmitted to everyone—patient, assistant or colleague—who came in contact with him.

It cannot be said that he was a prolific writer, but his occasional papers were carefully prepared, thoroughly thought out and bore conservative conclusions. A review of his publications over any given period reveals the fact that they deal chiefly with radiotherapy in some form—radium or roentgen rays. In the illustrations accompanying his papers on matters of diagnosis one recognizes the degree of care with which they were produced. His well known solicitude for the good results of technical painstaking, as thus shown, recalls to memory the pioneer day when consistent roentgenographic excellence, at a time when all contributory factors were inconstant and uncertain, meant patience and persistence—Byron Jackson's cardinal qualities.

Although in the fullness of a professional life so active there would appear to be no room for any salutary distracting influence, yet from the depths of an earnest religious conviction Doctor Jackson drew the spiritual stimulation of Scriptural study which reacted upon him in the manner of a genuine avocation. He was a profound analyst of the Bible, and for years had been widely known as an authoritative lecturer on the subject of that great literary classic. A mere cursory review of his career will show how consistently he applied its principles in the conduct of his daily life, to the utter benefit of his fellow-men. Many blessings were his reward—his home, his friends, his satisfying work with the ineffable gratification of accomplishment.

The record of Byron Jackson is a clear index of the happy trend toward a widespread distribution of better radiology throughout the United States. In this we have to thank the propitious Fates for such men as he who, as exemplars of fixed determination and individual effort amid dismaying circumstances, had the vision enabling them to point to opportunities still farther afield wherein others might as creditably employ the tools provided by Röntgen and the Curies with which to combat the ills of man.

For his notable part in this service to humanity, we respect the name of this kindly physician!

Percy Brown

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. B. Peirce, Royal Victoria Hospital, Montreal, Canada. Annual Meeting: Chicago, Ill., Sept. 19–22, 1939.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Next Annual Meeting: New York City, June 12, 1940.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: New City City, 1940.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Atlanta-Biltmore Hotel, Atlanta, Ga., Dec. 11–15, 1939.

RADIOLOGICAL SECTION, BALTIMORE CITY MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets twice annually in May and September.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. W. W. Furey, 6844 Oglesby Ave., Chicago.

RADIOLOGICAL SECTION, LOS ANGELES CO. MED. SOC.
Secretary, Dr. Wilbur Bailey, 2007 Wilshire Blvd., Los Angeles, Calif. Meets on second Wednesday of each month at County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. E. Mendelson, 120 E. 19th St., Brooklyn, N. Y. Meets monthly on first Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months, place of meeting selected by the host.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. C. J. Challenger, 3117 Logan Blvd. Meets second Thursday of each month October to May inclusive at the Hotel Sherman.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. H. A. Mahrer, 10515 Carnegie Ave. Meets at 6:30 P.M. at Mid-Day Club rooms on fourth Monday each month, October to April, inclusive.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. E. A. Schmidt, 4200 E. 9th Ave., Denver, Colo. Meets third Tuesday of each month.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. J. N. Moore, 210 Professional Bldg., Ocala, Florida. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. E. P. Halley, Decatur and Macon County Hospital, Decatur, Ill. Meetings held quarterly, time and place designated by president.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. C. C. Taylor, 23 E. Ohio St., Indianapolis, Ind. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. J. C. Bell, 402 Heyburn Bldg., Louisville. Meets annually in Louisville on third Sunday afternoon in April.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. C. S. Davenport, St. Lawrence Hospital, Lansing. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. I. I. Cowan, Mt. Sinai Hospital, Milwaukee, Wis. Meets monthly on first Friday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. J. P. Medelman, 572 Lowry Medical Arts Bldg., St. Paul.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets first Wednesday of each month, at 6 P.M., at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. A. O. Hampton, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. W. J. Marquis, 198 Clinton Ave, Newark. Meets annually at time and place of State Medical Society. Mid-year meetings at place designated by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. R. D. Duckworth, 170 Maple Ave., White Plains, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year—January, May, November.

PACIFIC ROENTGEN CLUB
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport, Pa. Annual meeting, June 2 and 3, 1939. Bedford Springs Hotel, Bedford, Pa.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. H. W. Jacox, 4800 Friendship Ave., Meetings held second Wednesday each month, 4:30 P.M., October to June at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. S. C. Davidson, 277 Alexander St., Meets on second Thursday from October to May, inclusive, 8 P.M., Rochester Academy of Medicine Building.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of October, January, March and May, at a place designated by the president.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco. Meets monthly on first Monday at 7:45 P.M., alternately at Toland Hall and Lane Hall.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

South Carolina X-Ray Society
Secretary, Dr. Hillyer Rudisill, Jr., Roper Hospital, Charleston. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg. Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. H. C. Harrell, 517 Pine St., Texarkana, Texas. Next regular meeting in Temple, Texas, 1939.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. V. W. Archer, University Hospital, University, Va. Meets annually in October.

Cuba

Sociedad Cubana de Radiologia y Fisioterapia
Secretary, Dr. Francisco Padron, Enrique, Villuendas 64, Havana, Cuba. Meets monthly in Havana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets monthly on third Thursday, from November to June inclusive, at 8:15 P.M., 32 Welbeck St., London.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine, 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. Barbara M. Key, 32 Welbeck St., London, W.1, England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to Nov. incl. for scientific discussion.

Canadian Association of Radiologists
Secretary, Dr. A. C. Singleton, Medical Arts Bldg., Toronto, 5, Ontario.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext., Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel. Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during month of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, II.52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent Secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Süd- und Westdeutsche Röntgensellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Röntgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the Spring, and one in the fall.

Societa Italiana Radiologia Medica
Secretary, M. Ponzio, University of Turin, Prof. Turin

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania.
Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly on first Monday at 8 o'clock.

Polish Society of Radiology
Secretary, Dr. Jan Kochanowski, 45 Gornoslazka St., Warsaw. Meets annually.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summertime.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime:

Society of Medical Radiology in Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen.
Meets the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Roentgen Society
Meets first Wednesday of each month, at 6:30 P.M. at Zentral-Röntgen Institut des allgemeinen Krankenhauses Alserstrasse 4.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University. Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyoto Japan. Meets bi-monthly on third Sunday.

PRELIMINARY PROGRAM OF THE FORTIETH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

September 19–22, 1939
Palmer House, Chicago, Illinois

12 O'CLOCK, NOON, SUNDAY, SEPTEMBER 17, 1939

Executive Council—Annual Meeting
Palmer House

MONDAY, SEPTEMBER 18, 1939

Willis F. Manges Trophy Golf Tournament
Flossmoor Country Club

TUESDAY, SEPTEMBER 19, 1939

Opening Session—8:30 A.M., Ball Room, Palmer House.

Call to Order, John W. Pierson, M.D., President.

Presentation of medals and certificates of merits awarded for Scientific Exhibits at the Thirty-Ninth Annual Meeting—President Pierson.

Address of Welcome—N. S. Davis, III, M.D., President of the Chicago Medical Society.

Installation of the President-Elect, Edward L. Jenkinson, M.D., Chicago, Ill.

Presidential Address.

9:15 A.M.

1. Clinical consideration of the stomach. Grant H. Laing, M.D., Chicago, Ill. (by invitation). (15 min.)
2. Varieties of ulcerative colitis: The diagnostic problem.
   (a) Clinical analysis. J. A. Bargen, M.D., Rochester, Minn. (by invitation). (15 min.)
   (b) Roentgenologic considerations. Harry Weber, M.D., Rochester, Minn. (15 min.)
3. Surgical treatment of ulcerating gastric lesions. Waltman Walters, M.D., Rochester, Minn. (by invitation). (15 min.)
4. The differential diagnosis of pyloric and prepyloric ulceration. Howard P. Doub, M.D., Detroit, Mich. (15 min.)
5. Syphilis of the stomach, with report of an unusual case. Clarence N. McPeak, Fitchburg, Mass. (15 min.)

   Discussion of Papers 1–5, inclusive, to be opened by H. E. Mock, M.D., Chicago, Ill. (by invitation), George W. Holmes, M.D., Boston, Mass., and B. R. Kirklin, M.D., Rochester, Minn. (20 min.)

11:05 A.M. Recess.

11:20 A.M.

6. Recent advances in roentgen examination of the neck; planigraphy of the larynx. Barton R. Young, M.D., Philadelphia, Pa. (15 min.)

   Discussion to be opened by W. Edward Chamberlain, M.D., Philadelphia, Pa. (5 min.)
7. A method for the measurement of the valvular efficiency of the heart. Ancel Keyes, M.D. (by invitation), H. L. Friedell, M.D. (by invitation) and Leo G. Rigler M.D., Minneapolis, Minn. (15 min.)

   Discussion to be opened by Wendell G. Scott, M.D., St. Louis, Mo. (by invitation). (5 min.)
8. Cineroentgenographic study of cardiac dynamics. William H. Stewart, M.D., New York, N.Y., and C. W. Breimer, M.D. (by invitation), New York, N.Y. (15 min.)

   Discussion to be opened by Merrill C. Sosman, M.D., Boston, Mass. (5 min.)
9. Body section roentgenography. Sherwood Moore, M.D., St. Louis, Mo. (15 min.)

   Discussion to be opened by Warren C. Breidenbach, M.D., Dayton, Ohio. (by invitation). (5 min.)
10. Relation of hormones to tumors of the female reproductive system. John I. Brewer M.D., Ph.D., and R. R. Greene, M.D. (by invitation), Chicago, Ill. (15 min.)

    Discussion to be opened by George H. Gardner, M.D., Chicago, Ill. (by invitation). (5 min.)

1:00 P.M. Recess.

2:00 to 4:30 P.M. Instruction Courses.* Seventh Floor.

4:30 P.M. Annual Executive Session of the Society. Ball Room.

* See June number for detailed description of Instruction Courses.

TUESDAY, 8:30 P.M., SEPTEMBER 19, 1939

Ball Room

Introduction—John W. Pierson, M.D. (President, 1938), Baltimore, Md.

The Caldwell Lecture "Roentgenology of Pancreatic Disease," James Thomas Case, M.D. Past President (1920) of the American Roentgen Ray Society, Professor of Radiology, Medical School, Northwestern University, Chicago, Ill.

Presentations—President Jenkinson.

WEDNESDAY, SEPTEMBER 20, 1939

8:30 A.M. Second Scientific Session, Ball Room.

11. Physical differences between types of penetrating radiation. Arthur H. Compton, Ph.D., Chicago, Ill. (by invitation). (15 min.)

12. Interstitial radium doses expressed in roentgens. Anna Hamann, M.D., Chicago, Ill. (by invitation). (20 min.)

13. Results of irradiation on primary bone tumors. Dallas B. Phemister, M.D. (by invitation) and Alexander Brunschwig, M.D. (by invitation), Chicago, Ill. (20 min.)

14. Effects of roentgen rays on normal brain tissue. Percival Bailey, M.D., Chicago, Ill. (by invitation). (20 min.)

15. Cancer and heredity. Maude Slye, Sc.D. (by invitation), Chicago, Ill. (20 min.)
Discussion on Papers 11–15, inclusive to be opened by Karl W. Stenstrom, Ph.D., Minneapolis, Minn., H. Dabney Kerr, M.D., Iowa City, Iowa, Merrill C. Sosman, M.D., Boston, Mass., Ira I. Kaplan, M.D., New York, N.Y. and Francis Carter Wood, M.D. (by invitation), New York, N.Y. (20 min.)

10:35 A.M. Recess

10:45 A.M.

16. Radiation therapy of inflammatory conditions. Arthur U. Desjardins, M.D., Rochester, Minn. (15 min.)
Discussion to be opened by B. H. Orndoff, M.D., Chicago, Ill. (5 min.)

17. Radiation therapy in cancer of the skin. Bernard P. Widmann, M.D., Philadelphia, Pa. (15 min.)
Discussion to be opened by John T. Murphy, M.D., Toledo, Ohio. (5 min.)

18. Late results in supervoltage roentgen therapy. (Comparative statistical analysis in selected groups). Traian Leucutia, M.D., Detroit, Mich. (15 min.)
Discussion to be opened by Richard Dresser, M.D., Boston, Mass. (5 min.)

19. The expression of roentgen-ray quality in the supervoltage range. Edith H. Quimby, M.S., L. D. Marinelli (by invitation), and T. R. Felsom (by invitation) New York, N.Y. (15 min.)

20. Further dosage measurements on 400 kv. roentgen-ray generators. Otto Glasser, Ph.D., Cleveland, Ohio. (15 min.)
Discussion of Papers 19 and 20 to be opened by Lauriston S. Taylor, Washington, D.C., and G. Failla, New York, N. Y. (15 min.)

21. The radiosensitive tumors. Ralston Paterson, M.D., Manchester, England (by invitation). (15 min.)
Discussion to be opened by Maurice Lenz, M.D., New York, N.Y. (15 min.)

1:00 P.M. Recess.

2:00 to 4:30 P.M. Instruction Courses. Seventh Floor.

THURSDAY, SEPTEMBER 21, 1939

8:30 A.M. Third Scientific Session. Ball Room.

22. Malignant tumors of the kidney in adults. Carl L. Gillies, M.D., Iowa City, Iowa. (15 min.)

23. Anomalies of the urinary tract. William J. Baker, M.D. (by invitation), Chicago, Ill. (15 min.)

24. Ureteral obstruction. B. H. Nichols, M.D., Cleveland, Ohio (15 min.)
Discussion of Papers 22–24, inclusive, to be opened by Hollis E. Potter, M.D., T. P. Grauer, M.D. (by invitation) and Herman Kretschmer, M.D. (by invitation), Chicago, Ill. (15 min.)

25. The treatment of far-advanced carcinoma of the breast. F. J. Hodges, M.D., W. S.

Peck, M.D. (by invitation), and Henry K. Ransom, M.D. (by invitation), Ann Arbor, Mich. (15 min.)
Discussion to be opened by U. V. Portmann, M.D., Cleveland, Ohio. (5 min.)

26. Radium treatment of vascular nevi. Ernst A. Pohle, M.D., Ph.D., Madison, Wis. (15 min.)
Discussion to be opened by Edward H. Skinner, M.D., Kansas City, Mo. (5 min.)

10:10 A.M. Recess.

10:20 A.M.

27. The comparative value of subarachnoid air versus lipiodol in the diagnosis of intraspinal protrusion of the intervertebral discs and of hypertrophy of the ligamentum flavum. John D. Camp, M.D., Rochester, Minn. (15 min.)
Discussion to be opened by W. E. Chamberlain, M.D., Philadelphia, Pa. (5 min.)

28. The roentgen findings in lower back and sciatic pain. Philip Lewin, M.D. (by invitation), Chicago, Ill. (15 min.)
Discussion to be opened by Claude Lambert, M.D. (by invitation), Chicago, Ill. (5 min.)

29. Chondrodystrophy calcificans congenita. Gerard Raap, M.D., Miami, Florida (15 min.)
Discussion to be opened by Edward C. Vogt, M.D., Waban, Mass. (5 min.)

30. Syphilis of the skeleton in early infancy; the nonspecificity of many of the roentgenographic changes. John Caffey, M.D. (by invitation), New York, N. Y. (15 min.)
Discussion to be opened by Ralph S. Bromer, M.D., Bryn Mawr, Pa. (5 min.)

31. Observations on circulatory changes in bone. F. A. Chandler, M.D. (by invitation) and John Norcross, M.D. (by invitation), Chicago, Ill. (15 min.)
Discussion to be opened by E. W. Ryerson, M.D. (by invitation), Chicago, Ill. (5 min.)

32. Chondro-osteodystrophy. Ralph S. Bromer, M.D., Bryn Mawr, Pa., and F. C. Hutton, M.D. (by invitation), Philadelphia, Pa. (15 min.)
Discussion to be opened by P. C. Hodges, M.D., Chicago, Ill. (5 min.)

33. Insufficiency fracture of the tibia resembling osteogenic sarcoma. George E. Pfahler, M.D., Philadelphia, Pa. (15 min.)
Discussion to be opened by Earl Barth, M.D. (by invitation) Chicago, Ill. (5 min.)

1:00 P.M. Recess.

2:00 to 4:30 P.M. Instruction Courses. Seventh Floor.

4:30 P.M. Executive Business Session of the Society. Ball Room.

THURSDAY, SEPTEMBER 21, 1939

7:00 P.M. GRAND BALL ROOM

Cocktails

The Annual Banquet
Presentation of the Gold Medal
of the Society

Dancing

FRIDAY, SEPTEMBER 22, 1939

8:30 A.M. Fourth Scientific Session. Ball Room.

34. Abnormally wide respiratory movement of the lower lung structures: Roentgen evidences of obstructive emphysema. Ross Golden, M.D., New York, N.Y. (15 min.)

35. Roentgenologic aspects of an epidemic of acute respiratory tract infection. Karl Kornblum, M.D., and Hobart S. Reimann, M.D., (by invitation), Philadelphia, Pa. (15 min.)

36. Non-putrid pulmonary suppuration. M. L. Sussman, M.D. (by invitation), New York, N.Y. (15 min.)

37. Basal exudates of subphrenic origin. L. R. Sante, M.D., St. Louis, Mo. (15 min.)

38. Primary carcinoma of the lung: a roentgenologic study of 225 cases. B. R. Kirklin, M.D., and John W. Olds, M.D. (by invitation), Rochester, Minn. (15 min.)

39. Recent progress in the bronchographic diagnosis of bronchogenic carcinoma. Pedro L. Fariñas, M.D., Havana, Cuba (15 min.)

Discussion on Papers 34–39, inclusive, to be opened by Edwin F. Hirsch, M.D., (by invitation), Chicago, Ill.; Lawrence Reynolds, M.D., Detroit, Mich., and Paul Holinger, M.D. (by invitation), Chicago, Ill. (20 min.)

10:20 A.M. Recess.

10:35 A.M.

40. Cystic disease of the lung. Carleton B. Peirce, M.D., Montreal, Canada (15 min.)

41. Visualization of the pulmonary circulation in lung disease. George P. Robb, M.D. (by invitation), Israel Steinberg, M.D. (by invitation), and Ursula J. Roche, M.D. (by invitation), New York, N.Y. (15 min.)

42. The value of chest roentgenograms in differentiating between primary and secondary polycythemia. Philip J. Hodes, M.D. (by invitation), and John Q. Griffith, M.D. (by invitation), Philadelphia, Pa. (15 min.)

43. Linear shadows in the lung (atelectasis, infarcts, interlobar fissures and inflammatory scars). Aubrey O. Hampton, M.D., Felix Fleischner, M.D. (by invitation), and Benjamin Castleman, M.D. (by invitation), Boston, Mass. (15 min.)

44. Prognosis in silicosis; roentgenologic study based on 500 cases. John T. Farrell, Jr., M.D., Martin J. Sokoloff, M.D., (by invitation), and Robert Charr, M.D. (by invitation), Philadelphia, Pa. (15 min.)

Discussion on Papers 40–44, inclusive, to be opened by George Scupham, M.D. (by invitation), G. de Takats, M.D. (by invitation), and Hollis E. Potter, M.D., Chicago, Ill. (20 min.)

45. The use of nitrites in cardiospasm. Max Ritvo, M.D., and Eugene J. McDonald, M.D. (by invitation), Boston, Mass. (15 min.)

Discussion to be opened by Sidney Portis, M.D. (by invitation), Chicago, Ill. and Richard A. Capps, M.D. (by invitation), Chicago, Ill. (5 min.)

46. The indications for the duration of irradiation in the treatment of cancer. Hermann Holthusen, M.D., Hamburg, Germany (by invitation) (25 min.)

Discussion to be opened by Edwin C. Ernst, M.D., St. Louis, Mo.

1:00 P.M. Adjournment

2.00 to 4:30 P.M. Instruction Courses. Seventh Floor.

MID-SUMMER RADIOLOGICAL CONFERENCE IN THE ROCKY MOUNTAINS

The following is a preliminary announcement and the guest speakers of the Mid-Summer Radiological Conference in the Rocky Mountains to be held in Denver, Colorado, on July 27, 28 and 29, 1939.

Hans A. Jarre, M.D., Detroit, Michigan.
James F. Kelley, M.D., Omaha, Nebraska.
Sherwood Moore, M.D., St. Louis, Missouri.
Bernard H. Nichols, M.D., Cleveland, Ohio.
Wendell G. Scott, M.D., St. Louis, Missouri.
Paul F. Titterington, M.D., St. Louis Missouri.
Edith H. Quimby, M.S., New York, New York.
Helen Quincy Woodard, M.D., New York, New York.
Symposium on the Radiation Therapy of Inflammations.
Symposium on the Radiation Therapy of the Diseases of the Blood and the Blood-Forming Organs.
Symposium on Urology.
Miscellaneous Subjects—Round-Table Discussions.

INSTITUTE FOR CONSIDERATION OF BLOOD AND BLOOD-FORMING ORGANS

The University of Wisconsin Medical School is to conduct an Institute for the Consideration of the Blood and Blood-Forming Organs, September 4–6, 1939. The program is to include papers and round-table discussions by European and American workers in the field of hematology. In addition to the discussions, the following formal papers are to be presented:

Dr. L. J. Whitts, Oxford, England, Anemias due to Iron Deficiency.
Dr. Cecil J. Watson, Minneapolis, The Porphyrins and Diseases of the Blood.

Dr. Cornelius P. Rhoads, New York, Aplastic Anemia.
Dr. E. Meulengracht, Copenhagen, Denmark, Some Etiological Factors in Pernicious Anemia and Related Macrocytic Anemias.
Dr. Harry Eagle, Baltimore, The Coagulation of Blood.
Dr. George R. Minot, Boston, Anemias of Nutritional Deficiency.
Dr. Russell L. Haden, Cleveland, The Nature of the Hemolytic Anemias.
Dr. Jacob Furth, New York, Experimental Leukemia.
Dr. Claude E. Forkner, New York, Monocytic Leukemia and Aleukocythemic Leukemia.
Dr. Edward B. Krumbhaar, Philadelphia, Hodgkin's Disease.
Dr. Louis K. Diamond, Boston, The Erythroblastic Anemias.
Dr. Edwin E. Osgood, Portland, Marrow Cultures.
Dr. Charles A. Doan, Columbus, The Reticulo-Endothelial System.
Prof. Hal Downey, Minneapolis, Infectious Mononucleosis.
Dr. Paul Reznikoff, New York, Polycythemia.

Physicians and others who are interested are cordially invited. A detailed program may be obtained by addressing Dr. Ovid O. Meyer, Chairman of Program Committee, University of Wisconsin Medical School, Madison, Wisconsin.

CHICAGO ROENTGEN SOCIETY

At a recent meeting of the Chicago Roentgen Society the following officers were elected: *President:* Dr. R. J. Maier; *Vice-President:* Dr. Adolph Hartung; *Secretary:* Dr. Chester J. Challenger. Meetings are held the second Thursday of each month from October to May, Hotel Sherman, Chicago.

INDIANA ROENTGEN SOCIETY

On May 21, 1939, the annual meeting of the Indiana Roentgen Society was held in Indianapolis. Dr. John D. Camp of Rochester, Minnesota, was a guest for the day and after a dinner in the evening read a paper on "Roentgenology of the Calvarium." Officers installed were: *President:* Dr. Juan Rodriguez, Fort Wayne; *President-Elect:* Dr. H. H. Inlow, Shelbyville; *Vice-President:* Dr. Wemple Dodds, Crawfordsville; *Secretary-Treasurer:* Dr. Clifford C. Taylor, Indianapolis.

LONG ISLAND RADIOLOGICAL SOCIETY

At the meeting of the Long Island Radiological Society held on May 26, 1939, the following officers were re-elected: *President:* Dr. S. G. Schenck; *Vice-President:* Dr. Koiransky; *Treasurer:* Dr. L. Goldfarb; *Secretary:* Dr. M. Wiener.

RADIOLOGICAL SOCIETY OF NEW JERSEY

At the meeting of the Radiological Society of New Jersey held in Atlantic City, June 7, 1939, the following officers were elected for the ensuing year: *President:* Dr. P. S. Avery, New Brunswick; *Vice-President:* Dr. J. G. Boyes, Plainfield; *Treasurer:* Dr. H. A. Vogel, Elizabeth; *Secretary:* Dr. W. James Marquis, Newark; *Counsellor:* Dr. A. W. Pigott, Skillman.

NEW YORK ROENTGEN SOCIETY

At a recent meeting of the New York Roentgen Society the following officers were elected for the ensuing year: *President:* Dr. Harry M. Imboden; *Vice-President:* Dr. Henry K. Taylor; *Secretary:* Dr. Roy D. Duckworth; *Treasurer:* Dr. Eric J. Ryan.

PACIFIC ROENTGEN CLUB

The annual meeting of the Pacific Roentgen Club was held at Del Monte, California, May 3, 1939. The following officers

were elected for the ensuing year: *Chairman:* Dr. Karl M. Bonoff, Los Angeles; *Members of the Executive Committee:* Drs. I. S. Ingber, A. C. Siefert and D. R. MacColl; *Secretary-Treasurer:* Dr. L. H. Garland.

PITTSBURGH ROENTGEN SOCIETY

At a recent meeting of the Pittsburgh Roentgen Society the following officers were elected: *President:* Dr. Zoe A. Johnston; *Vice-President:* Dr. Prentiss A. Brown; *Secretary-Treasurer:* Dr. Harold W. Jacox.

AMERICAN BOARD OF RADIOLOGY

The next examination of The American Board of Radiology will be held in Atlanta, Georgia, December 9, 10, and 11, 1939. Those wishing to receive appointments for this examination should have their applications in the office of the secretary not later than September 1. There will also be an examination in New York City in June, 1940, just preceding the meeting of the American Medical Association.

There will *not* be an examination in Chicago at the time of the meeting of the American Roentgen Ray Society.

B. R. Kirklin, *Secretary*

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers as space permits.

THE 1938 YEAR BOOK OF RADIOLOGY. Diagnosis: Edited by Charles A. Waters, M.D., Associate in Roentgenology, Johns Hopkins University; Assistant Visiting Roentgenologist, Johns Hopkins Hospital; Associate Editor; Whitmer B. Firor, M.D., Assistant in Roentgenology, Johns Hopkins University; Assistant in Roentgenology, Johns Hopkins Hospital. Therapeutics: Edited by Ira I. Kaplan, B.Sc., M.D., Director, Division of Cancer, Department of Hospitals, City of New York; Clinical Professor of Surgery, New York University Medical College, etc. Cloth. Price, $4.50. Pp. 512, with 511 illustrations. Chicago: The Year Book Publishers, Inc., 1938.

"The Life of Wilhelm Conrad Roentgen as Revealed in His Letters" is a welcome inclusion in this Year Book. This most interesting monograph, an inspiring literary production, first published in the September, 1937, issue of the *Scientific Monthly*, is reproduced almost in its entirety with illustrations from Dr. Glasser's private collection. Like many articles of interest to roentgenologists it was published in a journal not read by every reader of our official publications and would therefore have escaped the notice of many were it not for the valuable service rendered by Waters and Kaplan in giving it space in the Year Book.

Except for this innovation the 1938 Year Book follows the form established for its predecessors since 1932. As usual, the book is an accurate index of the progress made during the year both as to new discoveries and methods and summation and criticism of past experiences. Drs. Waters and Firor state that there is nothing sensationally new but call attention to many items of special interest such as a new technique for roentgenography of cartilages of the knee, arteriography in differentiation of bone tumors, cranial changes in pituitary dysplasias, recent advances in diagnosis of pulmonary diseases, articles on gastritis and cholecystography and newer contrast methods of injection of gas or air into the retromammary tissues and subarachnoid space, and lipiodol into the vagina.

They feel that Moore's improved laminagraph is the most important technical advance although it is too soon to evaluate the possibilities of the rotary kymograph.

An hour or two spent with the diagnostic section of the book will not only review one's reading of the year and refresh one's memory on known facts but reveal many that had escaped notice and will make the use of the book for daily reference easy and profitable.

Whether interested in therapy or not it behooves every roentgenologist to keep up with advances in this field. The common temptation is to pass over articles on treatment, leaving them for later reading or perhaps to be forgotten. With Kaplan's condensations one can keep up to date with minimal effort. And if a treatment problem arises one may find here the necessary facts as to management and prognosis. Most of the articles contain the essentials of the technique used and are therefore of practical help in daily usage.

Kaplan's appraisals of various articles are important because their substance is backed by an enormous experience and keen judgment. He agrees with Chamberlain and Young that not all tumors are best treated by the fractionated dosage of Coutard, and abstracts of several articles on both sides of this subject are given for the reader's guidance. Radiation biology and radiation physics claim a larger share of space than heretofore. Late reports from the work of the laborers in the search for the cause of cancer include experiments with hormones and estrogenic substances. Efforts to relate dosage of roentgen rays and radium in a common unit, comparative effects of 200 and 1,000 kv. roentgen rays, and biological action of neutron rays, are discussed in various abstracts.

Irradiation of rheumatic heart disease, bronchial asthma, arthritides, and inflammatory conditions throughout the body receives Dr. Kaplan's favorable comment. This

Year Book stands alone as a treasure house of current knowledge in the field of radiology. With its six predecessors it combines to form an invaluable compendium of reference material.

E. W. Hall

Digrafía: Nuevo metodo radiográfico para el estudio de los órganos móviles. By Guido Pollitzer. Paper. Pp. 104, with 41 illustrations. Buenos Aires: Imprenta Amorrortu, 1937.

Pollitzer describes and illustrates a device he brought out in 1936 for studying the various changes taking place in the outline of moving organs. He calls his process of roentgenologic study "diagraphy," distinguishes it from kymography and specifies the superior points of his procedure. To judge by the illustrations and text, diagraphy has so far found its greatest application in the study of the chest. The monograph has thirteen references to similar studies.

E. T. Leddy

A Handbook of Roentgen and Radium Therapy. By A. J. Delario, B.A., M.D., Radiologist, St. Joseph's Hospital, Paterson, N. J., and Community Hospital, Montclair, N. J. Cloth. Price, $8.00. Pp. 362, with numerous engravings, graphs and tables. Philadelphia: F. A. Davis Company, 1938.

In his preface the author states that he lays no claim to originality, that his book is, in fact, a collection of notes and charts mainly copied from the literature he has read. Unfortunately, the collection seems to have been made with little discrimination, and the author's own interpolations and explanations are incoherent and contain many errors.

Part I, entitled "A general discussion of roentgen and radium waves (sic!)," purports to contain basic physical and biological considerations. To one who does not already have a fair knowledge of radiological physics, that section would be difficult to follow. One wonders why he attempts to discuss such matters as spectral analysis, and omits any explanation of anything as fundamental in radiation therapy as the inverse square law. The biological chapters in this part contain a mixture of ideas, sometimes contradictory, gathered from various sources. One section must be condemned wholeheartedly by every radiologist; that is the series of diseases "usually reacting to" 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 15, and 20 threshold erythema doses. It is not stated upon what basis he has thus classified diseases, but it is obviously an extremely dangerous tabulation to publish. The same condemnation should be extended to his series of radiation prescriptions for the treatment of a large variety of tumors.

Part II contains a collection of 53 figures and 41 tables, with explanatory notes. For part of these, the source is given; some of the others are obviously taken from the literature but not acknowledged; some are apparently put together by the author on a basis of information obtained in his reading. At least half of the material in this section is of no practical importance at the present time. Some of the charts, such as the one purporting to show the percentage of radiation *absorbed by tissue* in therapy, are based upon erroneous physical concepts. Some which might be useful are so poorly explained as to be of little value. A list of errors in fact or interpretation found in even a casual inspection of this section would be lengthy.

Part III is about 140 pages of end-results of radiation therapy, taken from a wide range of sources and tabulated in various ways. This may contain historical material of value. However, judging by the standards of the first two parts, one would be dubious about accepting it without checking on its accuracy and completeness.

This is an expensive book and makes a good appearance. It is to be regretted that nothing favorable can be said of its contents. It is unfortunate that such a book should have been published without having first been submitted to competent criticism.

E. H. Quimby

Pyrétothérapie. Par Charles Richet, Professeur agrégé à la Faculté de Médicine de Paris, Médecin des Hôpitaux de Paris, Jean Surmont, Médicin Electro-radiologiste des Hôpitaux de Paris, et Pierre Le Gô, Electro-radiologiste des Asiles de la Seine. Paper. Price, 50 fr. Pp. 192, with illustrations. Paris: Masson et Cie, 1938.

This is the second book which has been presented to the medical profession in the field of artificial fever therapy. Neymann wrote a very comprehensive work a few years ago on this subject, and this book serves to complement

his work. Richet and his coworkers fail to be thoroughly convincing because they present a great deal of theory but very little is actual case results. Most of their experience has been with the use of antichancroidal vaccine rather than with physical agents in the production of artificial fever. They have not treated the variety of diseases nor the number of patients comparable to work in this country. The main subject matter is derived from a review of American literature. The first chapter covers the biological considerations, and the reaction of the organism to elevated temperatures. The second chapter enumerates the various methods employed in production of artificial fever. The final chapter discusses the clinical results and various diseases treated with artificially elevated body temperatures. Of particular interest is the statement that the employment of fever therapy should always be combined with chemotherapy in the treatment of syphilis. This is in direct keeping with the experience of fever therapists in this country. The book is a welcome addition to the rapidly accumulating literature on artificial fever therapy.

M. K. Newman

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 105 Rutledge Ave.
Charleston, S. C.

A DEVICE FOR MAINTAINING DEVELOPING SOLUTIONS AT A CONSTANT TEMPERATURE

By PAUL A. BISHOP, M.D., *and* GEORGE E. BIERCE, E.E.
From the Department of Radiology, Pennsylvania Hospital
PHILADELPHIA, PENNSYLVANIA

A CONSTANT, correct temperature of developing solutions has been difficult to maintain, in spite of adequate amounts of cold and warm water such as are generally available. The usual types of commercial mixing valves are unsatisfactory. They are not sufficiently accurate and usually do not compensate for variations of pressure and temperature at the incoming water lines, so that constant observation and regulation are necessary. Because of these shortcomings they cannot be trusted to remain in operation over night or week-ends and holidays. Resulting variations of solution temperature are not only annoying but also the cause of lack of uniformity of film quality.

To overcome these difficulties we developed a device that would keep the solutions at a constant temperature by continuous automatic operation, but with economical use of refrigerated water. This has been accomplished at a cost well below that of the usual commercial type of mixing valve. The device has been in uninterrupted operation for two years, during which time the solutions have remained constantly at 65° F., always ready for use even after week-ends and holidays. It is adaptable to any pre-existing supply of cold and warm water. Our refrigerating unit, which previously had been inadequate during the hot months, is now operating only part time during the day and runs only a few minutes at night and during week-ends. In addition to economy and convenience there has been a surprising improvement in uniformity of film quality.

DESCRIPTION

A special mercury bulb thermometer has been constructed for us, having three platinum contact points fused into the mercury tube. One is located close to the bulb so that it is always in contact with the mercury column. The other two are located at the 63° and 65° F. positions. These contacts are connected to wall mounted relays of high resistance, requiring but 2 ma. at low voltage. They permit so little current to pass through the thermometer that there is no arcing in the mercury tube.

The common contact point near the bulb and that at 65° F. are connected to the relay which controls a circuit to a magnetic or solenoid valve in the cold water line. The common and 63° F. points are connected with a similar relay which controls a circuit of the solenoid valve in the warm water line. The circuits are arranged so that both solenoid water valves are closed while the temperature is between 63° and 65° F.

The thermometer, protected by a perforated shield, remains immersed in the water bath surrounding the solution tanks. Some experimentation may be necessary to determine the most satisfactory position at which the thermometer should be fixed to give the desired temperature to the developer, since the temperature of the bath is not immediately uniform when hot or cold water enters it.

METHOD OF OPERATION

Assume that the temperature of the bath rises to 65° F. The mercury column in touching the contact at this point closes the circuit of the relay (*A*) on the cold water side (Fig. 1). The energized relay magnet draws a contactor (*B*) to it, closing the circuit of the magnetic valve (*C*) in the cold water line. The valve opens and allows sufficient cold water to flow into the bath to reduce the temperature below 65° F., at which point all circuits are broken and the cold water valve closed.

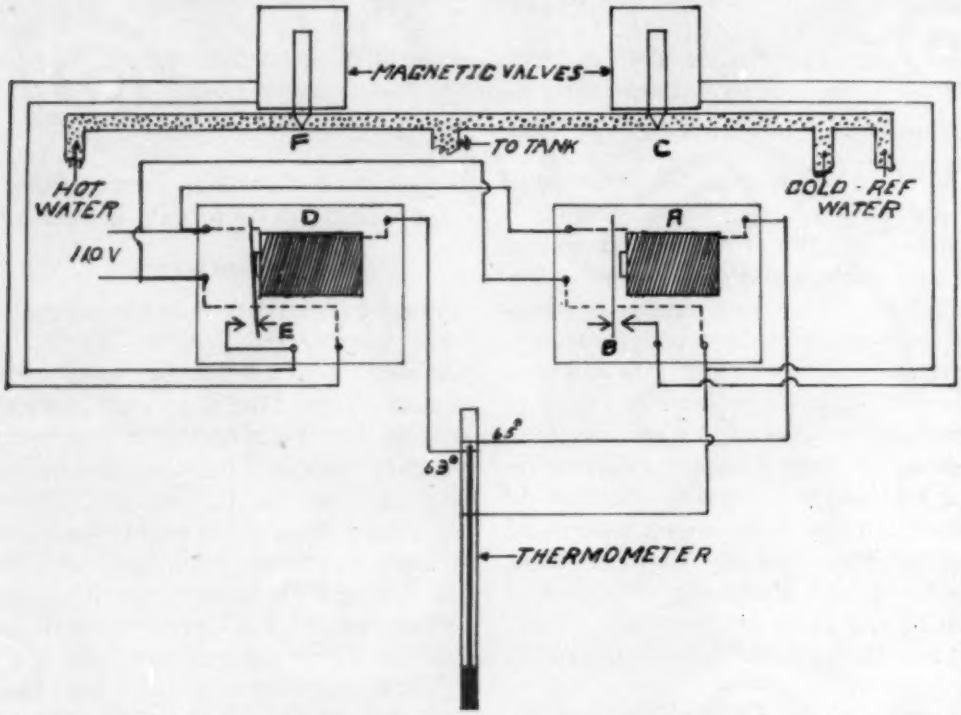


FIG. 1. Diagram showing apparatus with both valves closed, the temperature being between 63° and 65° F. See description in text.

If the temperature of the bath falls below 63° F., the mercury column goes below the contact at this point, breaking the circuit to the relay (*D*). This magnet thus releases a spring contactor (*E*) which closes the circuit of the magnet in the warm water valve (*F*), opening the valve and allowing warm water to flow into the bath until 63° is reached. At this point the relay circuit is again closed, the relay magnet energized, drawing the contactor to it, thus breaking the valve circuit and closing the warm water valve.

While this system theoretically permits a 2° fluctuation in the bath, such variations do not occur in actual use. There is a normal tendency toward an increased temperature of the developing solution, due to absorption of heat from the room, from films and hangers and from chemical activity during development of the films. Except while filling the tank the warm water valve is rarely called upon to function except under such unusual conditions as an abnormally cold room, due to open windows during the winter, or the cold water valve stuck in an open position.

The overflow from the bath should be drained through its own overflow pipe but can cascade into an adjacent washing compartment. The volume is small and the flow intermittent so that a separate supply of water, which can be of higher temperature, is necessary for washing.

Vol. 42, No. 1

STEREOROENTGENOSCOPY USING POLARIZATION

By WILLIAM SNOW, M.D.
Director Department of Roentgenology, Harlem and Bronx Hospitals
NEW YORK CITY

WE HAVE applied the principle of polarization of light in the construction of a stereoscopic roentgenoscope and caused experimental models to be made. This is in the form of a preliminary report.

PREVIOUS WORK

It has been the ambition of a number of men since the discovery of the roentgen rays to devise a stereoscopic roentgenoscope. The most outstanding workers were Caldwell, Snook and Kelly, Coolidge, Waite, and Rodriguez. The description of their apparatus can be found in the files of the United States Patent Office. Their basic principle was first brought out by Caldwell in 1902. The setup consisted of a double source of roentgen-ray energy about 2½ inches apart. A special double focus tube or two separate tubes could be activated by one transformer. The alternations of current would act first on one tube and then on the other. On the fluoroscopic screen two images of the part to be studied would obtain. Synchronously with the alternations of the current the viewer's right and then his left eye would be permitted to see the screen. This was done by rotating or vibrating some form of "blinker" before the eyes.

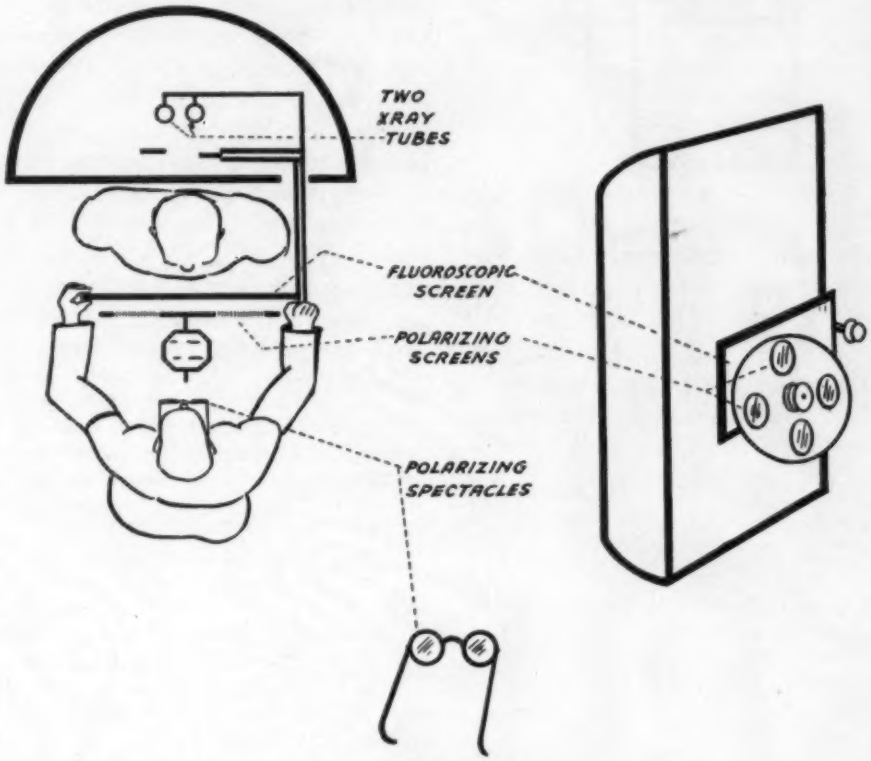


FIG. 1

DESCRIPTION OF APPARATUS

Our apparatus as in the past likewise consists of two roentgen tubes activated

by one transformer. In front of the fluoroscopic screen is caused to rotate a system of light polarizing screens by means of an 1,800 r.p.m. motor provided 60 cycle alternating current is used. The polarizers as they successively appear before the fluoroscopic screen polarize the light in the vertical and then in the horizontal plane. The observer wears spectacles of polarizing material vertical for one eye and horizontal for the other. The light will come through to the eye only when the polarizing screens are in the same phase (Fig. 1). This system has been used successfully by us in obtaining stereoscopic roentgenoscopy.

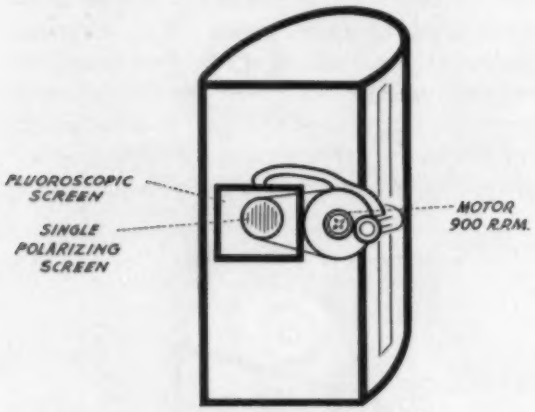


Fig. 2

It is advantageous to cut down the speed of the motor and at the same time increase the size of the polarizing field. To do this we have devised a system whereby a polarizing screen is mounted on bearings and caused to rotate on its own axis by a 900 r.p.m. motor (Fig. 2).

DISCUSSION

With the use of a Patterson B screen the apparatus worked well for thin parts, but for heavy parts the results were poor. Those who have used the previous types of stereoroentgenoscope have met with the same difficulty. It is our belief that this is undoubtedly due to lag image. With thin parts the lag image is, comparatively speaking, very faint, but with heavy parts vision is greatly reduced so that the lag image approaches the original image in intensity. If a fluoroscopic screen exists in which the lag image is properly eliminated, then our apparatus and the older ones will work well with heavy parts. We are therefore now working on plans to eliminate lag by attempting to discharge to ground the fluoroscopic screen between exposures of the two roentgen tubes.

We extend our appreciation and thanks to the Picker X-Ray Corporation for their advice and assistance in the construction of the apparatus.

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

King, G. C.: Encephalography in rapidly progressing cerebral atrophy due to trauma .. 146
Hauser, I. J., and Brownell, D. H.: Malignant neoplasms of the nasopharynx.... 146

Neck and Chest

Rosenblatt, M. B., and Bachman, A.: Upper lobe pneumonia in the adult..... 147
Schmitt, H.: Concerning esophageal ulcers.. 147
Johnston, L. M.: Congenital ciliated columnar epithelial cysts of the mediastinum.. 148
Pessin, S. B., and Pohle, E. A.: Hodgkin's disease with ulcerative involvement of the skin..... 148
Lynch, K. M.: Pulmonary asbestosis..... 149
Matz, P. B.: A study of silicosis..... 149
Klotz, M. O.: Association of silicosis and carcinoma of the lung..... 150
Habein, H. C., Miller, J. M., and Henthorne, J. C.: Tumors of pulmonary apices and adjacent regions involving the brachial plexus..... 150
Banyai, A. L.: Radiologic measurements of apico-basal relaxation of the lung during artificial pneumoperitoneum treatment.. 151
Andosca, J. B.: Contralateral spontaneous pneumothorax complicating artificial pneumothorax..... 151
Strongin, H.: Tension pneumothorax in newborn..... 151
Liljestrand, G., Lysholm, E., Nylin, G., and Zachrisson, C. G.: Normal heart volume in man..... 152
Maresh, M. M., and Washburn, A. H.: Size of the heart in healthy children..... 152
Iwasaki, H., and Shiga, T.: Roentgen kymographic examination of cardiac function.. 153
Appelbaum, E., and Kalkstein, M.: Coarction of the aorta..... 153
Denzer, B. S., and Horn, H.: Diffuse dilatation of pulmonary artery..... 154
Meyer, K. A., Hoffman, S. J., and Amtman, J. K.: Diaphragmatic hernia in newborn..... 154

Abdomen

Rhoads, J. E., and Stein, Eleanor: Splenectomy and ligation of gastric coronary vessels..... 154
Kaiser, R.: Addition to differential diagnosis between real and false diverticula of duodenum..... 155
Prevot, R.: Symptomless perforation of gastrointestinal tract..... 155
Pansdorf, H.: The visualization of deep-seated pathologic processes in the sigmoid in the roentgenogram..... 156
Werner, H.: Rapid functional test of the large intestine..... 156

Skeletal System

Sereghy, M.: The etiology of epiphyseolysis. 157
Law, J. L., and Perham, W. S.: Multiple cystic tuberculosis of the bones in children..... 157
Hodges, F. C.: Tuberculosis of the long bones 158
Sontag, L. W., and Harris, L. M.: Evidences of disturbed prenatal and neonatal growth in bones of infants aged one month. II. Contributing factors..... 158
Cornell, N. W., Bernheim, A. R., and Person, E. C.: Use of hydrochloric acid in certain cases of atrophy and delayed calcification in fractured bones..... 159

Blood and Lymph System

Dimtza, A., and Jaeger, W.: Indications for arteriography..... 159
Steiner, P. E.: Malignant lymphogranulomatous (Hodgkin's disease) cirrhosis of the liver..... 159

General

Goinard, P.: Practical observations on calcified hydatid cysts..... 160

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

KING, GEORGE C. Encephalography in rapidly progressing cerebral atrophy due to trauma. *Am. J. Dis. Child.*, Dec., 1938, *56*, 1330–1333.

Cortical atrophy, King states, is due to a disturbance in the cerebrospinal fluid-eliminating mechanism, which permits an increase in the amount of supracortical fluid with varying degrees of pressure. The factors which may cause the pathologic intracranial condition are many: encephalitis following measles, chicken pox or whooping cough; congenital malformation of the brain; congenital internal hydrocephalus; tumor of the brain; meningitis, or trauma. The first response of the brain to edema is an accumulation of fluid with resulting pressure and progressive atrophy and dilatation of the ventricles or the accessory channels. Obstruction at any point causes dilatation of the ventricular system and cortical shrinkage.

King reports a case which illustrates the rapidity with which cortical atrophy may develop. Trauma was the sole etiologic factor. The condition gave rise to a syndrome similar to that seen in cerebral lesions due to infection or in congenital malformations of the brain. Encephalograms on the first examination showed a normal ventricular system. Those made nine days later showed progressive atrophy of the right hemisphere and dilatation of the anterior horn of the right lateral ventricle. Examination by encephalogram thirty-five days later showed extensive dilatation of the ventricular system, especially of the right frontal horn and rapid progress of the atrophy. A last examination, fourteen months after the onset of symptoms showed still further dilatation of the anterior portion of the right frontal horn.

King thinks his observations in this case demonstrate the definite value of encephalography in determining the location and extent of intracranial damage. The condition revealed by the roentgen examinations was later confirmed by post-mortem examination. Young children tolerate encephalography rather well and symptoms of bradycardia and shallow respiration usually disappear in a day or two. The use of this procedure to appraise anatomic cranial defects is recommended.—*R. S. Bromer.*

HAUSER, I. JEROME, and BROWNELL, DURWIN H. Malignant neoplasms of the nasopharynx. *J. Am. M. Ass.*, Dec. 31, 1938, *111*, 2467–2473.

General practitioners, and even otolaryngologists, are still not well enough acquainted with malignant lesions of the nasopharynx to recognize them during their early stages. Carcinoma of the nasopharynx is a relatively common disease. All epithelial neoplasms arising from the lining membrane of the nasopharynx are medullary squamous cell carcinomas. In 50 cases in the department of otholaryngology at the University of Michigan Medical School during the past ten years confirmed by biopsy, 46 cases were called carcinoma, 3 were diagnosed as lymphoblastoma and in one instance the lesion was a myeloma.

Symptoms referable to the nasopharynx may be entirely absent. The initial complaints included swelling of the neck, aural pain, headaches or pain in the ear, pain in the throat, pain in the face, numbness of the face, facial paralysis, diplopia, nosebleeds, and nasal obstruction. Findings on objective examination included palpable nodes in the neck, discoloration of the tympanic membrane, fluid in the ear or a discharging ear, paralysis of the sixth nerve, hypesthesia, optic atrophy, paralysis of the superior oblique muscle, oculomotor paralysis, facial paralysis, drooping of the upper eyelid, vagus nerve paralysis, hypoglossal involvement, unilateral nerve deafness, Horner's syndrome and Gradenigo's syndrome. Roentgenograms of the skull were not taken as a routine diagnostic measure, although such films were made on many occasions and frequently showed involvement of the sphenoid sinus, invasion of the mastoid or petrous ridge or destruction of the base of the skull.

The authors believe that such terms as lympho-epithelioma and transitional cell carcinoma applied to malignant neoplasms arising from squamous epithelium are not necessary and that the group is adequately described by

the term medullary squamous cell carcinoma. They also believe that so-called lympho-epitheliomas and transitional cell carcinomas are highly anaplastic carcinomas. Carcinomas of the nasopharynx are radiosensitive because of their structure—no keratin, no cornification, very little cytoplasm and but little connective tissue between the cells.

The treatment of this disease is unsatisfactory. External irradiation of the local lesion is the usual procedure, from 200 to 400 roentgens daily through several portals, the dose being carried to skin tolerance, usually given over a period lasting from four to six weeks. In a few isolated cases following external irradiation, radon seeds were implanted in the local lesion. Yet, in spite of radiation therapy as intensive as it could be administered, only 3 patients of this series are free from carcinoma a year and a half after diagnosis. Even though these results have been most unsatisfactory, it appears that earlier diagnosis would result in a better prognosis.

(One of the discussants, Dr. James F. Percy, stated that he has treated a large number of late cases of malignant nasopharyngeal growths with the actual cautery, with a preliminary bilateral block dissection and ligation of the external carotid arteries by the method of Dawbarn. He has many of these cases, usually terminal, in which the patients are alive and free from morbidity years afterward.)—*S. G. Henderson.*

NECK AND CHEST

Rosenblatt, Milton B., and Bachman, Arnold. Upper lobe pneumonia in the adult. *Ann. Int. Med.*, April, 1938, *11*, 1845–1855.

Pneumonia of the upper lobe is not uncommon; it has been known for years but the diagnosis of it is infrequent. This is because physical signs are not striking when pathology is located in the upper lobes. Adams reported an incidence of upper lobe pneumonia of 18.5 per cent in his series of 522; MacCordick, 26 per cent; Warr and Alperin, 10 per cent. The authors found an incidence of 13.3 per cent among a group of 180. The age group ranged from the second to the ninth decades (though 70 per cent belonged to the third, fourth and fifth). The right upper lobe was involved three times as often as was the left. About half of the cases showed pneumococci (Types I–XXXII) but none were positive for the tubercle bacillus. Among those with pathology limited to the upper lobe, the mortality rate was 21 per cent; with multiple involvements, 70 per cent. The average mortality rate was 42 per cent. Most of these deaths occurred within the first ten days. The symptoms varied from insidious onset and gradual progression to the most abrupt onset of a meningitic sort. All cases showed a decided shift to the left by the Schilling count.

The treatment was chiefly supportive, forcing fluids on all and giving oxygen when necessary. Serum was given in only one case (because of the inability to obtain specific typings). About half of the cases were considered to be pulmonary tuberculosis on admission. Three typical cases are reported.—*A. A. de Lorimier.*

Schmitt, H. Zur Frage des Ulcus oesophagi. (Concerning esophageal ulcers.) *Röntgenpraxis*, May, 1938, *10*, 310–315.

Ulcer of the esophagus is a relatively rare disease. Pathologists have been acquainted with it for at least one hundred years. In autopsy material the lesion was found in 0.03 to 0.16 per cent of cases by various investigators. Among 4,000 esophagoscopic examinations, Chevalier Jackson found the condition in 21 instances.

The clinical picture of an ulcer closely simulates esophageal spasm, because the ulcer leads to severe and intermittent spasm. Dysphagia and vomiting are common and the symptoms are more marked after ingestion of solid foods than fluids. Cold fluids may lead to severe pain. Occasionally, however, an esophageal ulcer may be symptomless. Restrosternal pain is not a characteristic of this disease alone because it may occur in esophageal spasm also. Not rarely the symptoms are those of a gastric ulcer. If the pain is very severe, the patient eats sparingly and loses considerable weight. In fresh ulcers bleeding or perforation may occur.

The clinical study may cause the presence of an esophageal ulcer to be suspected, but it is the roentgenologic examination that establishes the diagnosis. Niches can be easily recognized during fluoroscopy and may be recorded with spot films. Superficial erosions, however, cannot be visualized roentgenologically.

Esophagoscopy is a valuable diagnostic aid, but a dangerous procedure in acute ulcers.

According to Roesler the following are the

contributing factors to the formation of peptic ulcers of the esophagus:

1. Primary changes in innervation due to constitutional impairment of the vegetative nervous system in the form of an esophageal spasm or cardiospasm.

2. Secondary spastic changes due to trauma, inflammatory processes, erosions of mucosa or as a distant action from gastric or duodenal ulcers.

3. Stasis of the contents due to circular or localized stenosing processes.

4. Impairment of circulation due to blood vessel spasm.

5. Action of inadequate stimuli on the esophageal mucosa.

The rôle of the gastric contents in the production of esophageal ulcer is much debated. It has been shown that gastric fluid is occasionally present in the distal esophagus due to aberrant gastric mucosal islands. On the other hand, the gastric juice may regurgitate into the esophagus.

The author reports a patient in whom a localized area of dilatation at the junction of the middle and distal thirds of the organ was found with localized spasm and small ulcer niche just distal to the dilatation. The patient had repeated hemoptysis, pain under the sternum and difficulty in swallowing.—*Eugene Freedman.*

JOHNSTON, LELAND M. Congenital ciliated columnar epithelial cysts of the mediastinum. *Am. J. Dis. Child.*, Aug., 1938, *56*, 313–320.

Johnston reports a case of congenital ciliated columnar epithelial cyst of the mediastinum in a girl, seven months of age. As is often the case, the roentgenogram in this patient showed no mass in the mediastinum but only atelectasis secondary to pressure on the air passages. The child died eight hours after admission to the hospital, before a complete examination, including laboratory procedures could be done. From the clinical findings it seemed most likely that there was a mediastinal mass pressing on the left bronchus and producing atelectasis of the left lung, in which there was a superimposed pneumonia. Autopsy added little to the diagnosis except to determine the type of mass in the mediastinum.

Dermoid cysts, teratomas and enlarged glands must be ruled out before a cyst of this type can be correctly diagnosed. Some of the cysts are accidentally found at autopsy in adults who have had no symptoms. In other instances, cysts of this type enlarge sufficiently to produce symptoms by pressure on the respiratory passages, as in the case reported. Case reports indicate that when once cysts become large enough to produce symptoms of pressure there is always a steady progression of these symptoms until operative intervention is attempted or until death results.

Probably the two most generally accepted theories of the bronchogenic origin of these cysts are: (1) During fetal life there is imperfect closure of the communication between the respiratory passages and the esophagus. Such a communication usually has its site above the bifurcation of the trachea, and formation of the cyst may occur when the two ends of the communication close and the passage itself remains. (2) The cyst represents a rest pinched off from the trachea or from one of the bronchi.—*R. S. Bromer.*

PESSIN, S. B., and POHLE, ERNST A. Hodgkin's disease with ulcerative involvement of the skin. *Am. J. Cancer*, Oct., 1938, *34*, 220–225.

This case report appears to be unusual in that a Hodgkin's primary lesion seems to have developed in the sternum, with extension to the skin and thereafter to regional lymphatics. The patient was a girl, aged fifteen, when first studied. In April, 1935, she complained of dull aching pain in the medial upper quadrant, left chest, with radiations of pain into her neck, left shoulder and upper arm. Occasionally she had a fever of 99 to 100° F. In July, 1935, a small lump developed over the second left costosternal junction. The leukocyte count was 9,500. Roentgenography of the chest was essentially negative. By October, 1935, roentgenography revealed a moth-eaten erosion of the lower left half of the manubrium. The leukocyte count was then 10,550. The lesion was curetted and found to consist of a grayish-yellow jelly-like tissue. Culture was positive for a non-hemolytic streptococcus. By March, 1936, this patient had to be readmitted because of a draining sinus at the site of the curettement. A debridement was performed, the microscopical study of which showed delicate granulation tissue infiltrated with lymphocytes, polymorphonuclear eosinophiles and neutrophiles. By June, 1936, a granulomatous mass had developed at the site of the surgical in-

cision; the skin was shiny red, and there were nodular tumefactions producing elevations as great as 3 cm. above the surface. Small lymph nodes could be palpated in the left supraclavicular and the left axillary regions. The leukocyte count was 20,550 (metamyelocytes, 1.3 per cent; stab forms, 40.3 per cent; segmented, 37.6; small lymphocytes, 7.3; large lymphocytes, 2.6; monocytes, 8.3; eosinophiles, 2.3 and basophiles, 0.3). The hemoglobin was 64 per cent; erythrocytes, 4,350,000. This picture suggested Hodgkin's disease and therefore a skin nodule was studied, which showed many eosinophiles and large cells which resembled Reed-Sternberg cells. Subsequently further adenopathy and mediastinal masses developed. A lymph node was removed from the right supraclavicular region; this revealed typical Hodgkin's lymphogranulomatosis.—*A. A. de Lorimier.*

Lynch, Kenneth M. Pulmonary asbestosis. iv. The asbestos body and similar objects in the lung. *J. Am. M. Ass.*, Dec. 11, 1937, *109*, 1974–1978.

One of the conspicuous and interesting features of the pulmonary condition resulting from the inhalation of asbestos dust by miners and mill workers in this material is the occurrence of certain curious bodies in the lungs and peribronchial lymph nodes and frequently in the sputum. These bodies measure from about 10 to more than 100 microns in length and from about 1 to 12 microns in thickness and are composed of a central translucent fiber or needle crystal, the asbestos crystal, with an enveloping shiny golden or brownish substance constructing various architectural figures. McDonald advanced the hypothesis that the asbestos body is a particle of asbestos fiber in the process of alteration and absorption by hydrolysis, with the silica passing into a colloidal state and a gel. Characteristically they are limited to the bronchial tract, the adjacent pulmonary alveoli, the peribronchial connective tissues and the peribronchial lymph nodes.

In the alveoli they are usually associated with or enclosed by phagocytes, mononuclear or multinuclear, some large foreign body giant cells occurring massed about those of large size. This may be the only cellular reaction to their presence. The presence of this material in abundance and over a long period does not necessarily result in fibrosis or other apparent damage to the tissue. Often, however, the asbestos bodies enter the tissues and apparently the lymphatics and lodge along the course, tending to stimulate growth of fibrous tissue about them. The disease is a diffuse type of fibrosis rather than the nodular form of silicosis proper.

The presence of these objects in the sputum indicates only inhalation of asbestos dust of sufficient duration for them to be formed. It is apparently possible to find them under conditions probably not conducive to disease. They may not be found even when the lungs are extremely disabled by fibrosis and the bodies are within the bronchioles and alveoli in large numbers.

After the discovery of the asbestos body the presence of such brownish objects within the lung came to signify practically a previous exposure to asbestos dust. This idea has been altered by the finding of similar bodies when no such exposure had occurred. Attention has been called to the occurrence of dust particles with brown coating in the lungs of workers in coal. Similar brown bodies have been observed in the lungs of a considerable number of subjects who died of heart failure, particularly rheumatism and arteriosclerosis. The author believes that chronic congestion of the lungs may play a part in the formation of some of these bodies. Usually, such deposits in the lungs may be distinguished from asbestos bodies, particularly because of the larger sizes and the translucent needle centrum of the latter. In the author's small series of silicosis cases the formation of an enveloping substance about the dust particles similar in color and appearance to that about asbestos and the other particulate matter here discussed was striking. The most common body is rounded, sometimes truly spherical, with a pale golden envelop on a black granule.

As to the nature of all of these objects, it appears that they are formed on a silica base, as is the asbestos body. On that hypothesis the group would become silica bodies, of which the asbestos body is a member.—*S. G. Henderson.*

Matz, Philip B. A study of silicosis. *Am. J. M. Sc.*, Oct., 1938, *196*, 548–559.

This study deals with 167 silicotic veterans, 120 of whom were living at the time of the report, 47 having died permitting 23 postmortem studies. The minimum age was thirty-seven; the maximum, sixty-five. One case

seemed to have acquired the condition as early as the age of twenty-three. The exposure time averaged 13.2 years though as short a while as one year appeared to be sufficient. Individual susceptibility seemed to vary depending upon pre-existing respiratory or sinus diseases, etc., and probably upon sensitivity to silica dusts, etc. The average duration of life after contact with the dusts (based on the 47 mentioned above) was 19.1 years; the minimum was eight years; maximum, fifty-one years.

Roentgenologically, these cases were grouped as: (1) first degree—having nodulations of 2 mm. or less; (2) second degree—nodulations 2 to 4 mm.; (3) third degree—nodulations 4 or more mm.; (4) conglomerate—with very large aggregations of nodules and interposed emphysema; (5) silicosis with infection—lesions indefinitely outlined with indications of secondary infection; (6) silicosis with tuberculosis; (7) silicotuberculosis (where lesions are so interwoven that one is unable to show separate background for either condition), and (7) asbestosis. Thirty per cent of these cases were considered 1st, 2nd or 3rd degree; 11 per cent, as silicosis with infection; 42 per cent, with tuberculosis; 16 per cent, as silicotuberculosis; and 1 case as asbestosis.

Clinically, the uncomplicated cases were manifested by dyspnea, cough, chest pain, impairment of breath sounds, fatigue, râles, and loss of weight. When tuberculosis complicated the condition, there were frequently added râles, excessive expectoration, loss of appetite and blood-tinged sputum. Roentgen criteria included accentuation of pulmonary markings, beadings, discrete nodule formations and widening of the mediastinal densities. Emphysema was evident in 42 per cent of the uncomplicated cases. When infection was added, there were mottlings, widening of the mediastinum, diffuse haziness extending inward from the lateral pleura, discrete and confluent nodular formations, etc. With tuberculosis there might be added fuzziness, perhaps caseation, pleurisy and cavitation.

The most frequent complications and coexisting diseases included pulmonary tuberculosis, cardiovascular disease, emphysema, other pulmonary infections, bronchiectasis, chronic pleurisy, chronic bronchitis and dental diseases.—*A. A. de Lorimier.*

KLOTZ, MAX O. The association of silicosis and carcinoma of the lung. *Am. J. Cancer*, Jan., 1939, *35*, 38–49.

The author merely mentions the high incidence of carcinoma of the lung among miners and the populations of Schneeberg and Joachimstal, saying that those cases most likely can be explained on the basis of radioactivity in the dust "as well as of the inhaled air." Various observers have noted a high incidence of dust contacts among the cases which developed carcinoma of the lung. Yet others such as Boyd, and Pancoast and Pendergrass have expressed opinions that the coexistence of these two lesions, namely, pneumoconiosis and primary carcinoma of the lung, was noted with extreme rarity.

There being such difference of opinion, the author analyzed 50 cases of silicosis which had come to post-mortem studies. Of these, 9 showed carcinoma in various organs—an incidence of 18 per cent (but this incidence compared with 17.7 per cent of a larger unselected series of 4,500 post-mortem studies). Of these 9, 4 were primarily bronchogenic (an incidence of 8.0 per cent as compared with only 1.17 per cent of the unselected series). These 4 cases are discussed in detail.

The usual symptomatology included dyspnea, cough, substernal pain, hemoptysis, and loss of weight and strength. The duration of these more aggravating symptoms varied from three weeks to four years. Two of the cases showed bloody fluid with thoracocentesis. The average age was forty-eight; 3 had been exposed to silica for more than twenty years (one, having had only five years' exposure, showed the highest percentage of silica in the lung tissue). All of the tumors were hilar in position.

The author believes that these relative statistics tend to indicate that silica may be a carcinogenic agent; just how, he would not say but considering the changes noted with dust experiments in animals he mentions chemical and physical irritation factors. This relationship might be indirect to conditions such as bronchiectasis, tuberculosis, interstitial pneumonia and lung abscess.—*A. A. de Lorimier.*

HABEIN, H. C., MILLER, J. M., and HENTHORNE, J. C. Tumors of the pulmonary apices and adjacent regions involving the brachial plexus. *Ann. Int. Med.*, April, 1938, *11*, 1806–1817.

Pancoast listed five leading criteria to characterize the "superior sulcus tumor": (1) roentgenologic evidence of a new growth at the apex of the lung; (2) homolateral pain referred along

the distribution of the involved nerves of the brachial plexus; (3) atrophy of the small muscles of the hand of the side affected; (4) early development of a homolateral Horner's syndrome; (5) roentgenologic evidence of destruction of adjacent ribs and vertebrae. Tumors of this location producing such evidence have been considered to be of pleural, branchiogenic, lymphatic, thymic and even of metastatic type.

Anatomically, explanation of these findings rests upon tumor developments in the fifth, sixth, seventh and eighth cervical and first thoracic segments of the spinal cord. By crossing the median cord of the brachial plexus (thereby affecting the ulnar nerve) or roots of its derivation (the eighth cervical and first thoracic), the small muscles of the hand are affected. Paralysis of the dilator fibers to the iris and of the superior tarsal muscle resulting in ptosis and paralysis of the retro-orbital muscle (resulting in enopthalmos) are produced by encroachment upon the cervical sympathetic chain. In like manner, there result decreased sweating over the region, hyperemia, and elevation of the cutaneous temperature.

Two cases are reported. The first one was ultimately found to be a carcinoma of the lung which had grown upward and mesially, deviating the thyroid gland to the opposite side and extending over the heart. There were numerous metastases. Biopsy of regional glands in the second case showed evidence of an adenocarcinoma. A summary of 27 other cases is presented. The author emphasizes the fact that the location of the lesion is the essential reason for the clinical picture but that no single type of neoplasm should be expected; even metastases to that site may be causative.—*A. A. de Lorimier.*

Banyai, Andrew L. Radiologic measurements of the apico-basal relaxation of the lung during artificial pneumoperitoneum treatment. *Am. J. M. Sc.*, Aug., 1938, *196*, 207–211.

Pneumoperitoneum has been advocated to provide relaxation and compression rest of the lung in cases of pulmonary tuberculosis—as a substitute measure in place of phrenic nerve surgery. The author has therefore studied 40 cases so treated, making measurements as to the elevation of the diaphragm. Five hundred to 1,000 cc. of air was injected at weekly and, later, two-week intervals. Roentgenograms were made at maximum inspiration and also maximum expiration, with the patient in the upright position. The apicobasal diameter was measured from the highest point of the apex of the lung to the highest point of the corresponding dome. Except where there were pathological changes, such as adhesions of the diaphragm to either the lung or the liver, there usually resulted a sustained elevation of the domes after repeated weekly injections. The maximum elevation during forced inspiration amounted to 7.3 cm.; during forced expiration, 6.4 cm.—greater than that observed after surgical paralysis of the phrenic nerve in a comparable group of patients.—*A. A. de Lorimier.*

Andosca, John B. Contralateral spontaneous pneumothorax complicating artificial pneumothorax. *Am. J. M. Sc.*, Oct., 1938, *196*, 559–564.

There have been very few cases reported where spontaneous pneumothorax has occurred during the course of artificial pneumothorax. Dunham reported it in 1926, at which time he was unable to find any previous mention of this complication in the literature. Burrell reported 3 such cases among a total of 10,889 punctures. The author checked the literature for the period 1933–1937, finding mention of only 2 cases. Pulmonary tuberculosis is probably the most frequent primary cause for spontaneous pneumothorax, though the direct cause may be rupture of an emphysematous bleb, perforation of a cavity or rupture of a subpleural tuberculous lesion. Probably because of increased effort and strain on the part of males, they seem to be affected about four times as often as females.

Two cases are reported. Clinically, the essential characteristics were sudden, sharp, agonizing pain, dyspnea, cyanosis, profuse sweating, fast thready pulse. Physically, there were the findings of displacement of the heart beat, hyperresonance and distant breath sounds. Confirmation of the diagnoses was gained by fluoroscopy and roentgenography. Both patients improved after aspiration but recurrences of the pneumothorax state followed.—*A. A. de Lorimier.*

Strongin, Harry. Tension pneumothorax in newborn. *Am. J. Dis. Child.*, July, 1938, *56*, 110–114.

Strongin reports a case of unilateral pneumothorax in the newborn, recognized four hours after birth. The recovery was spontaneous after

the simple withdrawal of air on two occasions during the first twenty-four hours of life. An unusual feature of the case was the presence of extensive subcutaneous emphysema, which disappeared promptly with the clinical improvement of the infant's condition. The roentgen examination of the chest showed a pneumothorax on the left side, severe subcutaneous emphysema, left side, and a shift of the mediastinum to the right.

Pneumothorax in the newborn usually results from trauma of birth; hence the study must extend over the first two or three weeks of life or longer. In any case of neonatal dyspnea, the chest should be examined both roentgenologically and fluoroscopically. In this way, many occurrences of the condition which are now overlooked would be recognized.—*R. S. Bromer.*

LILJESTRAND, G. LYSHOLM, E., NYLIN, G., and ZACHRISSON, C. G. The normal heart volume in man. *Am. Heart J.*, April, 1939, *17*, 406–415.

The authors point out that a false estimate of the volume of the heart may be obtained from anteroposterior orthodiagrams alone. They recommend making simultaneous posteroanterior and right lateral exposures and computing the volume on the principle of the Rohrer-Kahlstorf formula which is $V=0.63 \cdot F_a \cdot L_{max}$, where F_a is the area of the sagittal orthodiagram and L_{max} the maximum depth of the transverse orthodiagram. However, the authors make a correction for the enlargement of the shadow due to the projection on the film and a slightly different constant is employed. With the lateral exposure made at a target film distance of 150 cm. and the posteroanterior exposure at 200 cm., the formula becomes $V=0.38 \cdot l \cdot m \cdot n$. Where l and m are the axes of the ellipsoid in the frontal and n the axis in the lateral view. Double exposures in 10 cases showed a maximum inherent error of 4.7 per cent in the method. Measurements were made on 101 healthy men from twenty-one to forty-seven years of age divided into a younger and older group. The mean volume in the younger group was 700 cc. with the range from 430 to 970 cc. and in the older group 750 cc. for the average with the range from 550 to 1,000 cc. A better correlation was obtained with body surface than with body weight. The range of values for the cardiac volume in this normal group was 7.0–13.0 cc. per kilogram of body weight and 250 to 490 cc. per sq. m. of body surface.—*W. A. Evans, Jr.*

MARESH, MARION M., and WASHBURN, ALFRED H. Size of the heart in healthy children: roentgen measurements of the cardiac area and transverse diameter for sixty-seven children between birth and the age of six years. *Am. J. Dis. Child.*, July, 1938, *56*, 33–60.

The authors measured the cardiac area, total transverse diameter of the heart, internal diameter of the chest, clavicular-diaphragmatic distance and the surface area of the pulmonary fields on 1,026 roentgenograms of 67 children between birth and the age of six years four months. The height, weight, age and body surface area were recorded for each child, and a complete physical examination and electrocardiogram were made as well.

Curves which depict the growth in cardiac area and in cardiac transverse diameter, as noted in repeated examinations, showed rather marked variations in each child. The variations seen in the roentgenograms of different children were even more striking.

Maresh and Washburn discuss scatter charts of cardiac measurements plotted against height, weight, age, body surface area, internal diameter of the chest and the product of the body height and the internal diameter of the chest. They give correlation coefficients for these measurements. In general, the scatter is greater and the standard deviation larger with measurements of transverse diameter than those of cardiac area. They note, however, that the similarities in the growth curves for cardiac area and transverse diameter of the heart for the individual child suggest that undue emphasis may have been placed on the advantages of measuring cardiac area. Since the ratio $\frac{\text{internal diameter of chest}}{\text{total transverse diameter of heart}}$ is so commonly used as an estimate of the size of the heart, they give a table of values for the quotient for the age groups up to six years, calculated from their data.

In using Josephi's quotient of $\frac{\text{body surface area}}{\text{cardiac area}}$ on their data it was found that only 73.6 per cent of their values fell within Josephi's published range, 22.4 per cent being above his normal limits.

In using von Bernuth's quotient of

$\frac{\text{height} \times \text{internal diameter of chest}}{\text{cardiac area}}$ it was found that 93.5 per cent of their values fell within his normal range, with 3.7 per cent above and 2.7 per cent below.

They suggest wider normal ranges for Josephi's quotient, especially during the earlier years.—*R. S. Bromer.*

Iwasaki, H., and Shiga, T. Röntgenkymographische Untersuchung der Herztätigkeit. (Röntgen kymographic examination of cardiac function.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Nov., 1938, *58*, 484–507.

The authors review briefly the principles and precautions of the roentgen kymographic study of the heart. With special apparatus they study the roentgen kymogram to obtain (*a*) the diastolic and systolic transverse diameter of the heart, (*b*) the true amplitude of the excursions of the heart border at various points, (*c*) a computation of the changes in the surface area of the heart during the cardiac cycle, (*d*) the computation of various angles in the heart silhouette, (*e*) the type of wave (scissor shape, hack shape, trapezoid, pointed, rounded, small waved, dicrotic, or vascular type) and, (*f*) the time relationship of various motions. The findings in 110 normal hearts are described, together with the characteristic features of the kymograms in 154 cases of various pathological conditions affecting the heart. Of particular diagnostic significance is the concave (toward the inner side) aspect of the systolic arm of the wave in the left ventricular region which is pathognomonic of aortic stenosis. In decompensation, the heart is dilated with the transverse diameter increased in all segments, the amplitude of the waves is much reduced and they tend to be rounded in form and indistinct, particularly in the lower left border. In pericarditis there is a marked reduction of movement along both heart borders as in severe myocardial damage. Of unusual interest are the examples of beri-beri heart which are described and illustrated. Here the heart is dilated and the waves have a rounded form of diminished amplitude. These changes occur early in the disease and their recognition may lead to prevention of the late severe manifestations. The findings are particularly well shown in serial form during a study of experimental B_1 avitaminosis in a twenty-five year old male subject showing the development and cure of the disease. The last section deals with the findings in various arrhythmias and disturbances in the conduction system—*W. A. Evans, Jr.*

Appelbaum, Emanuel, and Kalkstein, Mennasch. Coarctation of the aorta. *Ann. Int. Med.*, Oct., 1938, *12*, 550–559.

This case of coarctation was recognized after special studies which were directed because of noting a brachial hypertension in a young man manifesting external anomalies (microcephalus and hemangioma). The authors emphasize the fact that this patient showed an impaired mentality in contrast to most others having this condition (normal or superior mentalities having been quoted and explained on the basis of increased circulation to the brain). A harsh systolic murmur was heard in the interscapular region and, following a split first sound, at the apex. The blood pressures were as follows: right arm, 180/130; left arm, 204/114; femoral pulsations were feeble on both sides. Roentgenologically there was evidence of enlargement of the ventricles; marked prominence of the region of the pulmonary artery, lack of visualization of the descending aorta in the left oblique, and subcostal notchings. The E.K.G. showed a left axis deviation, and deep Q waves in lead III.

The following physical signs are summarized as being characteristic: (1) normal or bounding pulses in the neck and upper extremities but subnormal or absent pulses in the lower extremities; (2) absence of a palpable abdominal pulsation but presence of an abnormal suprasternal thrust; (3) brachial artery hypertension with subnormal blood pressure in the popliteals, reversing the normal relative values; (4) compensatory collateral circulatory developments such as interscapular, suprascapular, axillary, precordial, internal mammary and epigastric enlargements—which might be conspicuous by harsh blowing systolic murmurs or by way of prominent pulsations which might even be palpated; (5) harsh systolic murmur especially along the left border of the sternum.

The roentgenological signs include: (1) bilateral erosion of the inferior borders of the ribs; (2) absence of the prominent aortic knob; (3) narrowing of, indentation, or gap in the descending portion of the aortic arch (left oblique view); (4) possibly dilatation of the ascending aorta.—*A. A. de Lorimier.*

DENZER, BERNARD S., and HORN, HENRY. Diffuse dilatation of the pulmonary artery. *Am. J. Dis. Child.*, Sept., 1938, *56*, 608–611.

Denzer and Horn report a case of diffuse dilatation of the pulmonary artery due to a patent ductus arteriosus and possibly associated with (rheumatic) mitral valvulitis. The recognition of this condition depends chiefly upon fluoroscopy. In their patient fluoroscopic examination showed a striking and definite pulsation in the upper hilar region of the right lung during each systole, and similar pulsations, though less striking and less diffuse, in the lower hilar areas on the right side. The pulsations were sudden and obvious and were more marked when the breath was held. The area of the upper pulsation was about the size of a 25 cent piece. In the midhilar region, pulsation of a similar mass was visible, and just below it a pulsation could be seen which was rather elongated and was directed toward the diaphragm. They noted, also, a relatively transparent area in the anteroposterior position between these pulsating bodies and the cardiac shadow proper.

They feel that until the frequency of this occurrence can be established by repeated fluoroscopic examination of large groups of children in whom increased pressure in the pulmonary arteries justifiably may be suspected, the clinical significance of the condition will remain problematic.

A loud machinery murmur was heard over the entire precordium but predominantly over the pulmonic area. It was also audible posteriorly. A thrill occupying the systole and part of the diastole was transmitted upward to the vessels of the neck and there was dullness in the second and third interspaces. Later two murmurs were heard at the apex, which seemed to be different in time from the loud pulmonic murmurs.—*R. S. Bromer.*

MEYER, KARL A., HOFFMAN, SAMUEL J., and AMTMAN, JENNIE K. Diaphragmatic hernia in the newborn. *Am. J. Dis. Child.*, Sept., 1938, *56*, 600–607.

The literature on diaphragmatic hernia is reviewed in this article and stress is placed on the importance of early diagnosis with timely surgical intervention. The authors report a case in which operation was successful thirteen days after birth. Roentgenograms taken seven days after birth showed loops of bowel in the left side of the chest which had displaced the lung and heart, forcing them to the right side. Barium fed the infant showed the stomach lying in the midline of the chest, protruding above the normal level of the diaphragm. Roentgen examination made two hours after operation showed the heart lying entirely on the right side of the chest. Four days after operation the heart showed less displacement and two months after operation both the heart and stomach lay in normal position.

Embryologic deficiency is commonly held accountable for congenital diaphragmatic hernia, which according to most authors usually occurs at the following sites: (1) through the hiatus pleuroperitonealis (foramen of Bochdalek) which is a gap in the musculature covered by pleura and peritoneum, and which is found posteriorly at the junction of the lumbar and costal portions of the diaphragm; (2) through the dome of the diaphragm (in cases of mild involvement not really a hernia but merely a bulging of part of the diaphragm known as "eventration"); (3) through the esophageal opening; (4) through one of the foramina of Morgagni; (5) through the gap left by the absence of the left half of the diaphragm, usually situated posteriorly.

Congenital diaphragmatic hernia, the authors state, is more common on the left side than on the right. The generally accepted reasons for this are: (1) the diaphragm is supported on the right by the mass of the liver; (2) the right crus is longer and thicker; (3) there are two fibrous bands on the right side.

When there is any suspicion of diaphragmatic hernia because of symptoms such as cyanosis, difficulty in feeding, vomiting or loss of weight, recourse should be had to roentgen studies. The treatment, the authors say, is surgical. Operative procedure is possible very early and is the only form of treatment when a portion of the intestine is in the thoracic cavity and certainly when signs of partial intestinal obstruction are present.—*R. S. Bromer.*

ABDOMEN

RHOADS, J. E., and STEIN, ELEANOR. Splenectomy and ligation of gastric coronary vessels; effect on esophageal varices in a case of Banti's syndrome with early hematemesis. *Am. J. Dis. Child.*, July, 1938, *56*, 119–125.

A case is reported of Banti's disease with esophageal varices and early hematemesis. At

the time the splenectomy was performed, the left gastric artery as well as the coronary vein was ligated. Roentgen examination of the esophagus before operation showed large esophageal varices. This finding was confirmed by esophagoscopy which revealed enormous dilatation of the veins. The latter projected into the lumen of the esophagus from all sides resembling earthworms. There was no ulceration or bleeding. After operation esophagoscopy showed dilated veins still present in the lower part of the esophagus but they were much less distended than before operation and no longer blocked the lumen of the esophagus.

Moyer examined 2 patients by esophagoscopy after splenectomy had been performed and found that varices persisted in both cases. Kirklin and Moersch had the opportunity to examine one patient with esophageal varices both before and after splenectomy and found no roentgen changes in the varices. Smith and Farber estimated that splenectomy reduces the load on the dilated veins at least 20 per cent, but stated that there is no evidence that the veins are reduced in size. Rhoads and Stein state that objective evidence that esophageal varices occurring in Banti's syndrome can be reduced in size by splenectomy and ligation of the gastric coronary vessels has not been presented. Such evidence was obtained in their case both endoscopically and roentgenographically. However, the surgical procedure differed from the usual in that the coronary artery of the stomach as well as the coronary vein was ligated in order to reduce further the load on the varicosed esophageal veins.—*R. S. Bromer.*

Kaiser, R. "Beitrag zur Differentialdiagnose echter und unechter Duodenaldivertikel." (Addition to the differential diagnosis between real and false diverticula of the duodenum.) *Röntgenpraxis*, Aug., 1937, *9*, 538–542.

Real diverticula of the duodenum are commonly observed. However, there is a group of bulgings of the wall of the duodenum which may imitate the appearance of diverticula, but are due to other causes. Herrnheiser reported the finding of a diverticulum-like structure of the descending part without evidence of a stenosis above or below the structure. The autopsy revealed a metastatic carcinoma in the pancreas which broke through the wall of the duodenum, producing an ulcerating area measuring about 4 cm. in diameter. Herrnheiser pointed out the difficulty in differentiating duodenal diverticula from ulcer craters or carcinomatous pseudo-diverticula. Clairmont and Schinz observed a diverticulum-like structure in the descending part which was due to an encapsulated abscess between the duodenum and liver, caused by a small perforated ulcer of the descending part.

The author observed a patient in whom the entire second portion of the duodenum was markedly narrowed. In the region of the ampulla the contrast material entered a cavity measuring about 2 cm. in diameter lying medially to the narrowed duodenum. The structure was sharply defined, tender on palpation and opened directly—without a connecting stem—into the duodenum. From a roentgenologic standpoint an ulcer, a real diverticulum and a necrotic carcinoma were considered.

The structure showed no peristaltic waves, it was fixed and was unchangeable in size on palpation. It showed no rugae. These signs ruled out a diverticulum. The sharp contour of the cavity and the smooth contour of the stenotic area were against a malignant lesion. The long clinical history was likewise in favor of a benign ulcer.

Autopsy revealed a benign ulcer just below the papilla of Vater with an increased connective tissue formation and with marked thickening of the muscularis in the region as well as in the neighborhood of the ulcer.—*Eugene Freedman.*

Prevot, R. Symptomlose Perforationen am Magen-Darmkanal. (Symptomless perforation of the gastrointestinal tract.) *Röntgenpraxis*, May, 1938, *10*, 303–307.

Acute perforations of ulcerative processes of the gastrointestinal tract are usually diagnosed clinically. The symptoms of beginning peritonitis are usually so alarming that there is no time for complicated diagnostic procedures. The roentgen method is of great value in differentiating this condition from acute gastritis or pancreatitis, but naturally only its positive findings are of importance. The diagnosis of perforation is established by finding free air under the diaphragm with the patient in the erect position. Even a minimal amount of air can be detected with this method.

The demonstration of spontaneous pneumoperitoneum, however, is not always identical with the clinical conception of perforation associated with beginning peritonitis. There are

cases reported where the roentgenologic sign of perforation was present without the clinical signs of peritonitis.

The author reports 2 patients who were admitted with upper abdominal pain of several days' duration. In both cases free air was found under the diaphragm. As the patients did not have clinical symptoms of peritonitis, conservative therapy was carried out. Both patients had a complete gastrointestinal study before discharge. One showed an ulcer crater on the duodenal bulb and the second one a large ulcer high on the lesser curvature side of the stomach. The author had an opportunity subsequently to observe 3 additional cases where similar clinical and roentgenologic findings were present.

In all these patients ulcerative lesions in the gastrointestinal tract were found, which were at the verge of perforation because their thin walls were already permeable to gases but not to fluids.

Reports of similar cases are rarely found in the roentgenologic literature, but are not uncommon in surgical journals.

In determining the therapeutic procedure in these cases the clinical findings are the important factors. The patients are to be kept under strict medical and surgical surveillance so that the correct procedure can be carried out as soon as symptoms of peritonitis present themselves.—*Eugene Freedman.*

Pansdorf, H. Die Darstellung tiefsitzender Krankheitsprozesse am Sigma im Röntgenbild. (The visualization of deep-seated pathologic processes in the sigmoid in the roentgenogram.) *Röntgenpraxis*, Nov., 1937, *9*, 764–769.

The transverse, descending, and ascending colon are easily visualized, but the sigmoid is difficult to visualize because its length and position vary greatly. It is often composed of a convolution of loops which lie in the small pelvis and are superimposed upon each other. If air is introduced after partial filling and if in addition the patient is turned, one is able to get a clear view of these loops. Schwarz recommends the moving of the roentgen tube toward the head to obtain a clear projection of the junction of rectum and sigmoid, which is a common site for cancers. Repeated examinations are often necessary to clear a difficult problem.

Despite all these methods, processes at the rectosigmoid junction may be missed. The author recommends an additional diagnostic aid. After the colon is completely filled with the enema, the patient's pelvis is elevated in the supine position with the hips widely separated. Furthermore, the patient is turned slightly to the left so as to project the ampulla free from the superimposed loops. The central ray is directed over the symphysis. On account of the high position of the pelvis, the superimposed loops move toward the head. The method has the advantage that in addition to the rectosigmoid junction, the remaining part of the sigmoid is also well visualized. After partial evacuation, air is injected into the rectosigmoid and subsequent roentgenograms are taken with the patient in the supine position, with the pelvis elevated.

The author shows illustrations demonstrating lesions in the rectosigmoid junction which were visualized only by this method.—*Eugene Freedman.*

Werner, H. Beschleunigte Functionsprüfung des Dickdarms. (Rapid functional test of the large intestine.) *Röntgenpraxis*, Oct., 1937, *9*, 683–689.

The examination of the colon with the aid of a contrast enema reached an exactitude in demonstrating wall changes as well as mucosal changes which cannot be approached by examination after oral examination of barium. Nevertheless, occasions arise when an examination of the function of the colon may be necessary to support a questionable local finding.

The usual examination of the function of the large intestine is a tedious procedure, lasting occasionally several days so that the evaluation of the individual phases and of their relationship is disturbed. Furthermore, the drying out of the contrast material creates unphysiologic conditions.

The author recommends a new method which combines functional and morphological features and is in many respects superior to the examination by contrast enema.

The patient receives a cleansing enema the day before as well as on the day of examination. At 9 A.M. the usual gastric examination is performed. Soon afterward a tablespoonful of Carlsbad salt dissolved in a glass of water is administered.

After such preparation the contrast material

reaches the cecum three to four hours after the administration of the contrast meal and at eight hours the entire large intestine becomes visualized. In such a manner the large intestine fills synchronously with the small intestine. The salt stimulates the peristaltic waves and forces the liquid small intestinal contents through the large intestines. The contrast material remains in liquid state while it passes through the colon and after defecation the mucosal relief becomes visualized.

This method is of invaluable aid in older individuals whose anal sphincter is insufficient and who are unable to hold the enema. In the presence of colitis the visualization of the mucosal pattern is often impossible after the enema, because the contrast material empties itself completely without coating the wall of the colon. The rapid peroral method allows, in many of such instances, the visualization of the mucosal relief.—*Eugene Freedman.*

SKELETAL SYSTEM

Sereghy, Michael. Daten zur Ätiologie der Epiphyseolyse. (The etiology of epiphyseolysis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, August, 1938, *58*, 129–136.

Diseases of the joints may be divided into two groups, those affecting primarily the synovia, cartilage, or capsular ligaments and those affecting primarily the bone adjacent to the joint. In this second group belong Perthes' disease and coxa vara adolescentium which have many similarities. Both are diseases of childhood affecting boys more often than girls and usually between the age of five and twelve. In Perthes' disease the softening and destruction of the bone take place in the head of the femur, while in coxa vara adolescentium the destruction occurs distal to the epiphyseal line in the middle of the neck.

Schulz of Vienna in 1913 was one of the first to show that coxa vara occurs in dystrophia adiposogenitalis, and Schmidt was able to show that 15 per cent of boys with coxa vara exhibited an outspoken adiposogenitalis. Mushrooming of the femoral head and disturbances in the femoral neck have also been reported in adults showing sexual hypofunction and other endocrine disturbances. Two additional cases are reported by the author. Both had an active epiphyseolysis unrelated to trauma and asociated with a dystrophia adiposogenitalis. Treatment in such cases must include not only an attack on the local orthopedic problem, but also an attempt to supply the whole organism with the necessary vitamins and hormones.—*W. A. Evans, Jr.*

Law, John L., and Perham, W. S. Multiple cystic tuberculosis of the bones in children. *Am. J. Dis. Child.*, Oct., 1938, *56*, 831–845.

Three factors probably predicate the rarity of cystic tuberculosis of the diaphysis of long bones. After an infection of the blood stream, localization of the tubercle bacilli in the bones elects the metaphyses owing to the anatomic situation of the end arteries; the caseous process of a tuberculous lesion of bone is more rapid usually than fibrosis and may lead to abscess or sinus formation; generalized tuberculous infections in children are apt to precipitate a fatal outcome in a relatively short time.

The authors report a case of osteitis tuberculosa multiplex cystica, as described by Jüngling in adolescents and in adults. Their patient was, however, a girl, six years old. The history, physical findings, results of laboratory studies, the picture shown by serial roentgenograms and the progress of the disease are given in detail. The long bones and also the small bones of the hands and the feet were involved. They conclude that the disease may occur in children as well as in adults. The presence of cutaneous lesions is not necessary for the diagnosis. Such lesions are usually not present in children, or they may appear only after the disease has been present for several years. The pathologic process is an entity with varying degrees of involvement and destruction of bone. The differences in the degrees of osseous involvement does not necessitate nomenclature other than mention of the anatomic location or than such a designation as disseminated cystic tuberculosis of the bones. The cystic changes may involve only the small bones of the hands and feet, only the long hollow bones or both. Classification according to Jüngling's description, in which only the hands or the feet are affected, or, with restriction to Kienböck's description, in which only the long hollow bones are involved, is not necessary, as the disease process has the same cause in both instances.

The primary focus of tuberculous infection in nearly all cases is probably intrathoracic. The roentgenologic appearance of the osseous lesions is reasonably characteristic and is of distinct diagnostic value. Law and Perham

believe that differential diagnosis of cystic osseous lesions of doubtful causation should include tuberculosis, syphilis, osteomyelitis, xanthomas, hyperparathyroidism, multiple myeloma, coccidioidal granuloma, leprosy and enchondroma.—*R. S. Bromer.*

HODGES, FRANK C. Tuberculosis of the long bones. *J. Bone & Joint Surg.*, Jan., 1939, *21*, 148–153.

Hodges reports 6 cases of tuberculosis of the long bones. The condition is infrequent, but the diagnosis is made easier by recalling its clinical manifestations from time to time. It must be suspected clinically before the diagnosis can be established by more extensive examinations and study. His cases demonstrate the accepted opinion that tuberculosis is a rapidly fatal disease when accompanied by active lesions in the lungs and the bones. Lymphadenopathy was present in 3 of the patients and biopsy demonstrated it was due to tuberculosis. A progressive tuberculous bone lesion induces osteoporosis, due to disuse and the increased vascularity which accompanies it. If the tissue defense is adequate to produce a local fibrous reaction, the circulation may be diminished, with consequent osteosclerosis. This type of reaction is seen in the more chronic types of tuberculous bone lesions. When such an osteosclerotic lesion is located in a cancellous bone, it stands out in marked contrast to the osteoporosis of the surrounding bone. The various degrees of tissue resistance may be visualized roentgenographically. When resistance is great, there is lamellar osteosclerosis; when resistance is less, there is lamellar osteoporosis and possibly bone cysts.

Sequestration does occur in tuberculous osteomyelitis and, as always, the sequestrum appears in the roentgenogram as an area of increased density, which is in contrast to the decreased density of the surrounding bone. Sequestration is produced by sudden stoppage of the blood supply to the area by the inflammatory process. In obscure bone lesions, such as may be produced by tuberculosis, biopsy and guinea-pig inoculation should always be employed. The cases are reported in detail with reproductions of roentgenograms illustrating the various lesions.—*R. S. Bromer.*

SONTAG, LESTER W., and HARRIS, LOUISE M. Evidences of disturbed prenatal and neonatal growth in bones of infants aged one month. II. Contributing factors. *Am. J. Dis. Child.*, Dec., 1938, *56*, 1248–1255.

In a previous communication, Sontag described the appearance and incidence of circular striae in the tarsal bones of infants at the age of one month. These striae appeared in lateral roentgenograms of the foot as fine white lines, usually from 0.5 to 1 mm. inside the margin of the calcaneus and of the talus. In a few instances there were present about the periphery solid bands or thickened temporary zones of calcification which later became resolved into a stria. In the present paper, the authors compared the characteristics of pregnancies and deliveries of mothers whose infants showed these tarsal striae at one month of age. The progress of these infants from birth to one month was also compared. The pregnancies of the mothers of the infants with and without striae differed appreciably in the following respects:

1. The incidence of persistent nausea and vomiting was much greater in the mothers of the infants with striae.

2. All mothers in poor health, other than that occasioned by persistent nausea and vomiting, gave birth to infants in whom striae developed.

3. In the infants of these mothers whose economic status was unusually low, striae developed.

4. The mothers of the infants with striae had an appreciably higher weight when not pregnant than did the mothers of the infants without striae.

Deliveries of the infants with and without striae differed as follows:

1. A greater proportion of the infants with striae were first born.

2. The incidence of forceps delivery was much greater among the infants with striae.

3. A greater percentage of the infants with striae were born of precipitate labor.

Other factors, such as the mother's basal metabolic rate, calcium content of the serum and hemoglobin content, the length of labor, the weight of the infant at birth and the gain in weight from birth to the age of one month, were not appreciably different for the two groups.

Sontag and Harris conclude that the tarsal striae are the result of disturbances in growth produced by the process of birth itself and in-

fluenced by such factors as maternal health and nutrition. They believe that the shock of birth is an important factor and that it is determined by the severity of the birth process plus the physical condition of the infant. They consider the mechanism comparable to that involved when striae are laid down in the long bones of growing children, as a result of a surgical procedure or of severe illness.—*R. S. Bromer.*

Cornell, Nelson W., Bernheim, Alice R., and Person, E. Cooper. The use of hydrochloric acid in certain cases of atrophy and delayed calcification in fractured bones. *J. Bone & Joint Surg.*, Jan., 1939, *21*, 40–50.

The authors observed a reduced gastric acidity and decrease in the volume of gastric contents in 8 cases of fractured bones in which excessive bone atrophy and delayed calcification were found about the sites of fracture. Their findings and experience in these cases lead them to conclude that the addition of hydrochloric acid (4 to 8 cc. of a 10 per cent solution, three times a day) to a diet high in calcium and vitamins increases the absorption of calcium and furthers the calcification of bone. Five of such cases were treated.

They based their procedure on the following considerations: Absorption of calcium is promoted by factors which tend to keep calcium in solution in the intestine, and vice versa. Calcium salts are soluble in acid and relatively insoluble in an alkaline medium. Intestinal acidity is due to hydrochloric acid from the stomach, to fatty acids formed during digestion, and to lactic acid fermentation. Vitamin D is also associated with the production of intestinal acidity. A diet high in calcium and vitamins is used as a part of their usual treatment of fractures, not only because of the general good effects of a diet enriched by these substances, but also to increase the amount of "available" calcium in the blood.

Attention is called to the fact that the use of hydrochloric acid without the proper calcium intake may be harmful, since hydrochloric acid besides furthering the utilization of calcium also increases its excretion. Their schedule of treatment, diets, etc., is given in full.—*R. S. Bromer.*

BLOOD AND LYMPH SYSTEM

Dimtza, A., and Jaeger, W. Über die Indikation der Arteriographie. (The indications for arteriography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, July, 1938, *58*, 40–45.

The authors recommend the use of arteriography when the disturbance in circulation is so marked as to raise the question of amputation. In this way one gains an idea of the extent of the disease and the state of collateral circulation. They confirm the observation of Demel and Sgalitzer that the intra-arterial injection of the opaque material also has a definite therapeutic value. In doubtful cases the distinction between endangiitis obliterans and a vasoneurotic symptom-complex can be made only by arteriography. Arteriography has also proved helpful in medicolegal cases with unexplained pain in an extremity.

In 70 percutaneous injections, para-arterial injections occurred 6 times, and in 1 case there was nausea and fever. The authors use thorotrast and have not observed any damage to the arterial wall.

Five cases are described and illustrated in which arteriography was helpful—one with Bürger's disease which was much improved by lumbar ganglionectomy, one with advanced arteriosclerosis in the legs, but no arterial obstruction, one with arteriosclerotic obliteration of the femoral artery and an extensive collateral circulation, one with a post-traumatic angiospastic condition in one finger, where the arteriography revealed the basis for pain, and one case with advanced endangiitis requiring amputation of one leg and ganglionectomy which has benefited the other.—*W. A. Evans, Jr.*

Steiner, Paul E. Malignant lymphogranulomatous (Hodgkin's disease) cirrhosis of the liver. *Am. J. Path.*, Jan., 1937, *13*, 109–119.

Hodgkin's infiltration may produce symptoms by its bulk when it is cellular and by its contraction when it is scirrhous. The natural course of lymphogranulomatous tissue leads to fibrosis. Irradiation accelerates this process.

A case of Hodgkin's disease is reported where a male, aged fifty-five, had noted swellings of superficial lymphatics for about two years. The diagnosis had been proven about three months before his death. Roentgen therapy had been given to the superficial glandular enlargements with spectacular involution of them. However, jaundice, ascites, rectal bleeding and anemia developed and the patient died. Postmortem studies revealed residual lymphatic en-

largements along the left side of the pelvis and along the left side of the aorta. These were very scirrhous, showing only small foci of lymphogranulomatous tissue. These regions had not been irradiated; therefore this change appeared to represent "spontaneous fibrosis." The spleen and vertebrae also showed involvement. The liver showed monolobular cirrhosis; cellular granulomatous foci (showing features of Hodgkin's) were found scattered along periportal regions. Some of these were barely discernible because of a greater amount of fibrosis while other areas were simply fibrotic.—*A. A. de Lorimier.*

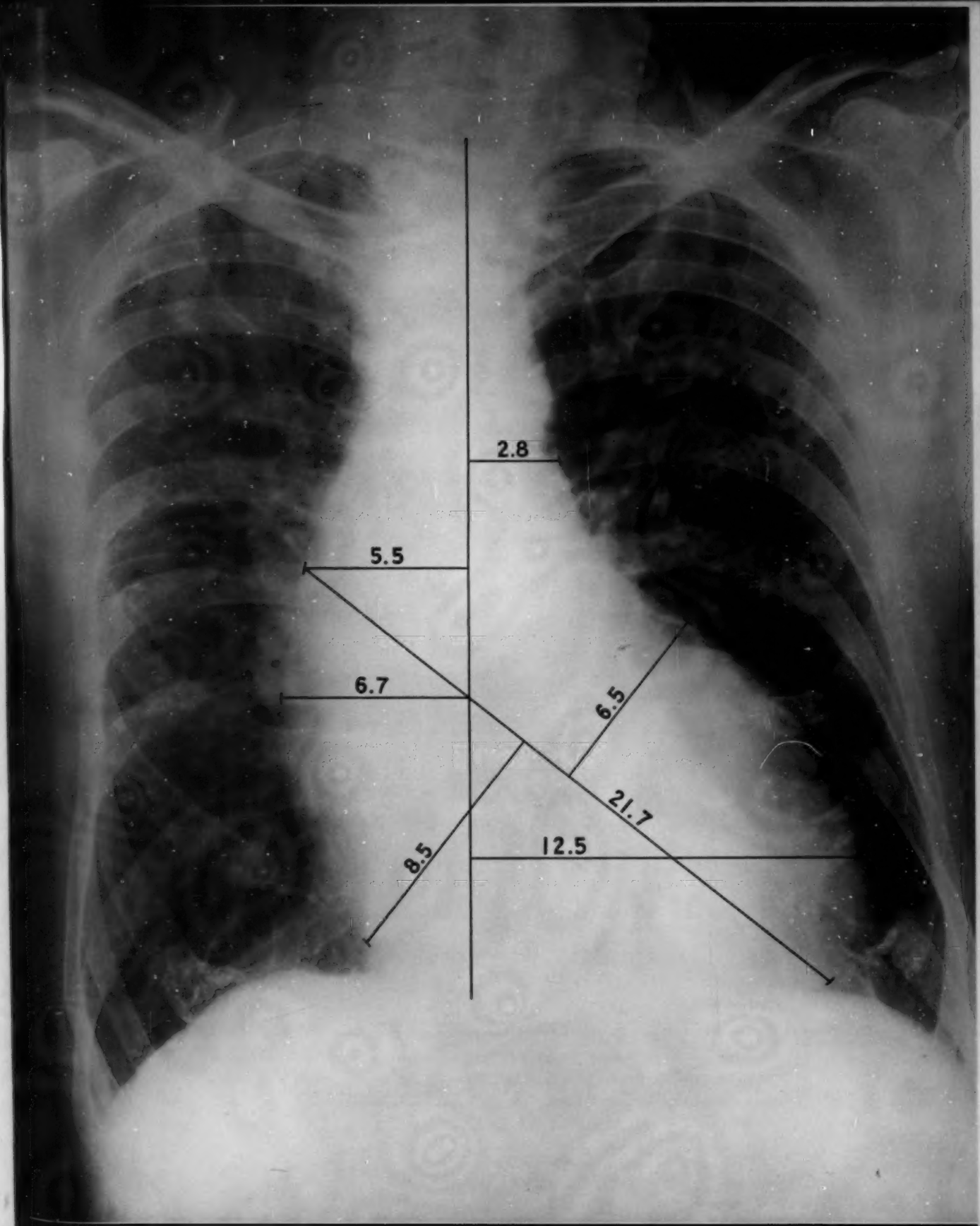
GENERAL

GOINARD, PIERRE. Quelques notions pratiques sur les kystes hydatiques calcifiés. (Practical observations on calcified hydatid cysts.) *Presse méd.*, June 25, 1938, *46*, 1023–1025.

Calcified cysts are more common than is thought. The frequency of calcification varies according to organ studied, age of patient and age of the cyst. It is most frequent in liver, spleen, kidney, muscle and subpleural tissue; it is rare in the lung. Echinococcus disease is one of young people; three-fourths of the patients with hydatid cysts are less than forty years of age; two-thirds of patients whose cysts are calcified are over forty. In general, for the same organ, cysts whose walls are calcified are apt to be found in an age group thirty years older than the non-calcified cysts.

Clinically, calcified cysts are very variable. They may be an accidental finding; they may open into adjacent structures and drain like a bone infection with sequestration.

Roentgenographic findings are very important but are not pathognomonic. Differentiation may have to be made from costal calcification, biliary calcification, glands, chronic abscess, tumor, non-parasitic cyst, hematoma, etc. Where the calcium is diffuse, thick and almost homogeneous, particularly if its outlines are irregular and its wall broken or retracted, the cyst is apt to be in advanced involution, but this is only a probability and positive dog inoculation may be obtained. Calcified cysts usually can be watched and need not be operated on. Cysts which show little calcification usually require operation. Complicated cysts which, for example, open into a viscera, require intervention, particularly because the calcified wall makes the lesion less yielding.—*M. L. Sussman.*



Reproduced from a radiograph made on

EASTMAN ULTRA-SPEED SAFETY X-RAY FILM

(Technical Factors on Back of This Page)

Technical Factors for Radiograph Reproduced on Reverse Side of this Page . . . .

REGION	**Thorax (heart)**
POSTURE	**Erect**
PROJECTION	**Postero-anterior**
THICKNESS	**25 cm.**
ANODE-FILM DISTANCE	**72″**
TUBE FOCAL SPOT	**2 mm.**
TUBE FILTER	**.3 mm.**
FILM	**Eastman *Ultra-Speed***
SCREENS	**Eastman *High-Definition***
KV.P.	**78**
MA.S.	**18**
DEVELOPMENT	**Kodalk X-ray Developer (3½ min., 65° F.)**
FIXATION	**Eastman X-ray Fixer**

Eastman *Ultra-Speed* X-ray Film . . . Eastman *High-Definition* Intensifying Screens . . . Kodalk X-ray Developer Powders . . . Eastman Prepared X-ray Fixing Powders are made to complement each other. This combination in your laboratory will provide the best assurance you can buy of diagnostically excellent radiographs.

Be sure to visit the Kodak Building at the New York World's Fair—One Hundred Years of Photography . . . Cavalcade of Color . . . Kodak in Medicine, Science, Education. Photographic experts to assist you. You'll find this a genuinely worth-while exhibit.

EASTMAN KODAK COMPANY

Medical Division—Rochester, N. Y.

(See reverse side of this page)